CONTENTS

PART I

THE STUDY OF THE RHEUMATIC DISEASES

CHAPTER	PAGE
1 INTRODUCTION TO ARTHRITIS AND THE RHEUMATIC DISEASES (J L. HOLLANDER)	15
	16
	17
	19
	21
	22
	25
	27
	28
	30
	40
3 THE STRUCTURE AND FUNCTION OF ARTICULAR TISSUES (J L HOLLANDER)	42
4 THE SYNOVIAL FLUID (H C COGGESHALL)	48
5 THE SEDIMENTATION RATE (H C COGGESHALL)	56
6 RADIOLOGY IN RHEUMATIC DISEASES (C J SMYTH)	64
Rheumatoid Arthritis	67
Degenerative Joint Disease—Osteoarthritis	80
"Mixed Arthritis"	84
Arthritis Due to Specific Infection	85
Arthritis Due to Mycotic Disease	94
Miscellaneous Conditions	95
Hemophilic Arthritis	98
Traumatic Arthritis	99
Gout	102
Roentgen Therapy in Rheumatic Diseases	106
Radioactive Substances in Treatment	113
7 ORGANIZATION OF AN ARTHRITIS CLINIC (WALLACE GRAHAM)	118

PART II

RHEUMATOID ARTHRITIS

8. THE ETIOLOGY OF RHEUMATOID ARTHRITIS (CHARLES RAGAN)	123
9 THE PATHOLOGY OF RHEUMATOID ARTHRITIS (E. F. ROSENBERG)	
Articular Lesions	130
Pathology of Subcutaneous Nodules	139
Striated Musculature	141
Peripheral Nerves	142
The Integument	143
Central Nervous System	144
Tendons, Tendon Sheaths and Bursae	144

CHAPTER — PAGE

10. 148
150
154
156
Prognosis 160
Atypical Rheumatoid Arthritis 160
Rheumatoid Arthritis in Children (Still's Disease) 161
Felty's Syndrome 162
Sjogren's Disease and Rheumatoid Arthritis 162

11. PSORIASIS AND RHEUMATOID ARTHRITIS (WALLACE GRAHAM) 164

12 THE VISCERAL LESIONS OF RHEUMATOID ARTHRITIS (E F. ROSENBERG)
Cardiac Lesions 171
Pulmonary Lesions 177
Hepatic Lesions 177
Lymph Nodes 179
Other Visceral Changes 179
Ocular Lesions 182
Causes of Death in Rheumatoid Arthritis 183

13 LABORATORY FINDINGS IN RHEUMATOID ARTHRITIS (H. C COGGESHALL) 186

14 RHEUMATOID ARTHRITIS. DIFFERENTIAL DIAGNOSIS (H C. COGGESHALL) 199

15 T 224
229
243
245
249

16. G 251
252
254
261
268
278
279
285

17. RG) 288
290
295
297
300
301
311
312
315
316

18. HYDROCORTISONE (J L HOLLANDER) 319
321
322
323
325
327

19. PREVENTION OF DEFORMITIES FROM ARTHRITIS (J. G. KUHNS AND T. A. POTTER) 337
Posture 338
Splinting of Affected Joints 343
Methods for Preventing Deformity 350

COMROE'S

ARTHRITIS

and Allied Conditions

Completely Revised and Rewritten.

EDITOR

JOSEPH LEE HOLLANDER, M.D.,

COLLABORATING EDITORS

Nathan R. Abrams, M.D.
Victor G. Balboni, M.D.
Edward W. Boland, M.D.
Ralph H. Boots, M.D.
Morris A. Bowie, M.D.
Howard C. Coggeshall, M.D.
Richard H. Freyberg, M.D.
Wallace Graham, M.D.
W. Paul Holbrook, M.D.
John G. Kuhns, M.D.
John Lansbury, M.D.
Theodore A. Potter, M.D.
Charles Ragan, M.D.
Edward F. Rosenberg, M.D.
Charley J. Smyth, M.D.
Philip D. Wilson, M.D.
Philip D. Wilson, Jr., M.D.

FIFTH EDITION, WITH 199 ILLUSTRATIONS

LEA & FEBIGER
PHILADELPHIA
1953

CHAPTER PAGE

20. CORRECTION OF ARTHRITIC DEFORMITIES (J. G. KUHNS AND T. A POTTER) 370
    Manipulation of Deformed Joints 373
    Orthopedic Surgery in Rheumatoid Arthritis 373
    Orthopedic Treatment of Osteoarthritis 385
    Correction of Deformities in Specific Joints 388

21. 405
    406
    409
    418
    420
    428
    429
    443
    Exercises for the Arthritic Patient 449
    Miscellaneous Physical Measures Sometimes Used in Arthritis 454

22. OCCUPATIONAL THERAPY (M A. BOWIE) 465

23. REHABILITATION OF THE ARTHRITIC PATIENT (M A BOWIE) 473

24. SPA THERAPY (J. L. HOLLANDER) 487

PART III

FACTORS INFLUENCING THE ONSET OR COURSE OF THE RHEUMATIC DISEASES

25. W BOLAND) 497
    497
    499
    500

26. FOCAL INFECTION IN RHEUMATOID ARTHRITIS (WALLACE GRAHAM) 509

27. CLIMATE AND THE RHEUMATIC DISEASES (W P HOLBROOK) 517

28. THE RÔLE OF THE ENDOCRINE GLANDS IN RHEUMATIC DISEASES (JOHN LANSBURY)
    The Pituitary Gland and Arthritis 522
    The Adrenal Glands and Arthritis 523
    The Thyroid Gland and Arthritis 526
    Rheumatism and the Menopause 526
    The Parathyroid Glands 529
    The Prostate Gland 530

29.
    532
    533
    535
    538
    539

30. DIET AND VITAMINS IN ARTHRITIS (E F ROSENBERG) 542

PART IV

IMPORTANT RHEUMATIC DISEASES CLOSELY ALLIED TO RHEUMATOID ARTHRITIS

31. SPONDYLITIS (E W. BOLAND)
    Rheumatoid (Ankylosing) Spondylitis 547

PRINTED IN U. S. A.

CONTENTS

PAGE

[illegible] Holbrook

570
572
576

579
583
585
588
590
593
594
601
605
614

623

629

[illegible] Rheumatism) (Wallace

635

PART V

THE DISEASES OF COLLAGEN

649
653
655
659

[illegible] John Lansbury)

670
681
685

PART VI

DEGENERATIVE JOINT DISEASE (OSTEOARTHRITIS)

[illegible] of Degenerative Joint Disease

691
694

[illegible] of Degenerative Joint Disease

708

[illegible] Joint Disease (N[illegible]brams)

PART VII

[illegible] FROM DISORDERS [illegible]BOLISM

PREFACE

and corticotropin (ACTH) on rheumatic diseases was announced just prior to the publication of the Fourth Edition in 1949. Many parts of this book

of this book, add new chapters, and completely revise it to bring it up to date. In order to prevent this Fifth Edition from becoming out-dated even as it is issued, each contributing editor has not only kept himself aware of developments as reported in the literature, and at important medical meetings, but also has partaken actively in the research in his field, and has corresponded extensively with others working on related problems. Many recent developments in Rheumatology are reported in this volume even before reports have appeared in the medical literature, either based on the author's own research, or work he is sure will stand the critical test of time. Opinions are expressed not with the "voice of authority," but are based on long experience and critical study.

The contributors are all "specialists" in the Rheumatic Diseases, either internists or orthopedic surgeons particularly interested in arthritis, drawn from many sections of the United States and one from Canada. Most of them conduct arthritis clinics and are teachers in this field Since almost half of the editors worked together in the Rheumatic Disease Centers of the Army during the War period, the lessons there learned in treating the thousands of arthritis patients are incorporated in this volume

All the collaborators are active members of the American Rheumatism Association, the national society for dissemination of knowledge in this field, and are active in working for the Arthritis and Rheumatism Foundation in its efforts to advance the knowledge and provide better care for the victims of arthritis

This volume deals with the entire field of the Rheumatic Diseases Any disorder producing pain and stiffness of the locomotor system might be confused with rheumatoid arthritis. For this reason many clinical entities seemingly far removed from arthritis have been discussed, such as sarcoidosis, scleroderma, bone tumors, and many of the local disorders of the musculo-skeletal system *The field of Rheumatology cannot be confined* to Arthritis alone, but should be considered as the study of Medicine from the viewpoint of one particularly interested in diseases of the connective tissue system It is impossible for the physician to render efficient service to the rheumatic patient unless he acquires at least a working knowledge of *all* the painful disorders of the musculo-skeletal system, which will then permit him to make an accurate diagnosis

CHAPTER PAGE

42 OTHER FORMS OF METABOLIC ARTHRITIS (J. L. HOLLANDER) 775

PART VIII

ARTHRITIS CAUSED BY OR OCCURRING WITH SPECIFIC INFECTIONS

43. GONORRHEAL ARTHRITIS (V G BALBONI) 779

44 REITER'S SYNDROME (INFECTIOUS URO-ARTHRITIS) (J L HOLLANDER) 794

45 TUBERCULOUS ARTHRITIS (V. G BALBONI) 813

46 SYPHILITIC JOINT DISEASE AND NEUROGENIC ARTHROPATHY (V. G BALBONI) 831

47 [illegible]

842
847
850
851

48 ARTHRITIS WITH TROPICAL DISEASES (V G BALBONI) 866

PART IX

MISCELLANEOUS CONDITIONS OF OBSCURE ETIOLOGY

49 HEMOPHILIC ARTHRITIS (M A BOWIE) 877

50 SARCOIDOSIS (JOHN LANSBURY) 885

51 TUMORS OF JOINTS AND TENDON SHEATHS (J. G KUHNS AND T A POTTER) 890

PART X

REGIONAL DISORDERS OF JOINTS AND RELATED STRUCTURES

52 TRAUMATIC ARTHRITIS AND ALLIED CONDITIONS (N R ABRAMS)
- Traumatic Arthritis 907
- Sprains 912
- Temporomandibular Joint Syndrome 918
- Wounds of Joints 919
- [illegible] 921
- [illegible] 921
- [illegible] 923
- Osteochondritis Dissecans 923
- Synovial Cysts 925
- Pellegrini-Stieda Syndrome 926
- Ganglion 926
- Peritendinitis Crepitans 927
- Injuries to Muscles and Tendons 927

53 INTERNAL DERANGEMENT OF THE KNEE JOINT (N R. ABRAMS) 933

54 [illegible]

944
945
960
963
965

The arrangement of sections and chapters has been extensively changed from the last edition, in order to bring discussions of related conditions closer together. An effort has been made to prevent unnecessary repetition of material, and much old material has been deleted either because it was redundant or obsolete Nearly every chapter in this book has been rewritten at least in part, and 93 new illustrations have been added or substituted Entirely new chapters have been added on Corticotropin, Cortisone, and Hydrocortisone, Intra-articular Injection of Hydrocortisone; and Low Back Pain and Sciatica. Completely rewritten are the chapters on Etiology of Rheumatoid Arthritis, Focal Infection, Endocrine Glands, Pregnancy and Jaundice Effect, Diet and Vitamins, Non-articular Rheumatism (Fibrositis), and Dupuytren's Contracture. Fifteen other chapters (*e g.*, The Collagen Diseases) have been so extensively rewritten that they too are almost new chapters Only the extensive deletion of old material has prevented this volume from becoming much larger than the previous edition.

Most physicians use a text such as this primarily as a reference work, and for this reason the summaries in box form have been retained and increased to be readily available for those who have too limited time for reading an entire chapter The extensive references to the literature have been retained and brought up to date for those desiring further detail. It is hoped that this Fifth Edition will be of interest not only to the general practitioner, but also to the internist, pathologist, dermatologist, industrial surgeon, physician in the Armed Forces, orthopedic surgeon, physiatrist, [illegible]

to whom the editors make grateful acknowledgement. They also thank the authors and publishers of many recent articles and monographs for permission to reproduce material and illustrations The publishers have been most cooperative and helpful, and have again given the editors a free hand in the preparation of this Fifth Edition. Drs. Ernest M. Brown, Jr., Louis Udell, Ralph A Jessar, and James O. Crittenden have given generously of their time assisting in proof reading and indexing this volume.

PHILADELPHIA, PA J. I

CHAPTER PAGE

Syphilitic Spondylitis 570
Osteoarthritic (Hypertrophic) Spondylitis 572
So-called Infectious Spondylitis 576

32. RHEUMATIC FEVER (W P HOLBROOK)
Incidence 579
Etiology and Pathogenesis 583
Pathology 585
Laboratory Findings 588
Relationship to Other Diseases 590
Differential Diagnosis 593
Symptoms 594
Course, Prognosis, and End Results 601
Treatment 605
Prevention 614

33 PALINDROMIC RHEUMATISM (E F. ROSENBERG) 623

34. INTERMITTENT HYDRARTHROSIS (CHARLES RAGAN) 629

35. FIBROSITIS SYNDROME (NON-ARTICULAR RHEUMATISM) (WALLACE GRAHAM) 635

PART V

THE DISEASES OF COLLAGEN

36 THE COLLAGEN DISEASES (JOHN LANSBURY) 649
Serum Sickness 653
Periarteritis Nodosa 655
Disseminated Lupus Erythematosus 659

37 THE SCLERODERMA GROUP OF COLLAGEN DISEASES (JOHN LANSBURY)
Scleroderma 670
Dermatomyositis 681
Calcinosis 685

PART VI

DEGENERATIVE JOINT DISEASE (OSTEOARTHRITIS)

38. THE ETIOLOGY AND PATHOGENESIS OF DEGENERATIVE JOINT DISEASE (N R ABRAMS) 691
Normal Aging of Joints and Pathogenesis 694

39 SYMPTOMS AND LABORATORY FINDINGS OF DEGENERATIVE JOINT DISEASE (N. R. ABRAMS) 708

40. *TREATMENT OF DEGENERATIVE JOINT DISEASE* (N R ABRAMS) 728

PART VII

ARTHRITIS FROM DISORDERS OF METABOLISM

41. GOUT (C. J. SMYTH) 739
Etiology and Pathogenesis 741
Uric Acid Metabolism 745
Pathology 750
Clinical Course 753
Diagnosis 758
Provocative Factors 761
Treatment 762
Prognosis 770

FOREWORD

Bernard Isaac Comroe graduated with the highest honors from the Medical School of the University of Pennsylvania in 1929 and was the winner of three of the chief prizes After an interneship at the University Hospital of his Alma Mater he joined the faculty of his school and soon became recognized as an inspiring teacher, an excellent clinician, and a man of high integrity and unbounded energy Early in his career he developed a sympathetic interest in the pathetic patients with arthritis for whom so [illegible]

[illegible] classes of students. In both of these activities he was highly successful.

After some years of study of arthritis he realized as everyone did, that there was no adequate text on arthritis, and so, as a labor of love, and from a sense of duty, he labored assiduously to produce in 1940 his book "Arthritis and Allied Conditions." This work at once achieved wide acclaim and became the outstanding volume on this subject A second edition soon followed and in 1944 a third edition, thus testifying to the worth and popularity of the book

Doctor Comroe died in 1945 just as he was beginning to reap the well-deserved honors and rewards for his achievements. His death was a truly great loss to Medicine, to the field of Arthritis, to his students, and to his friends.

It is a gratifying testimony to the continued value of Comroe's Arthritis that it has been used as the basis for revisions by a group of the leaders in the field of Arthritis under the editorial leadership of Dr. Joseph L Hollander. Doctor Comroe would have been the first to wish the work kept up-to-date and to appreciate the fact that frequent revisions are made necessary by the rapid advances of knowledge in the subject. He would have been quick to admit that the field has widened until today no book on this subject could be the work of one individual He would be proud could he but know of the calibre of the group of cooperating authors The Fourth Edition, produced by this group in 1949, was so successful that this (Fifth) edition was soon planned and now has been produced

Doctor Comroe honored me in 1940 by dedicating his book to me, and it is with real sincerity that I write these few words about him and the book which is his lasting memorial At the same time I wish to say that the present revision is a worthy successor to the earlier editions and I prophesy its equal success

O H. PERRY PEPPER, M.D

ARTHRITIS

PART I

THE STUDY OF THE RHEUMATIC DISEASES

CHAPTER 1

INTRODUCTION TO ARTHRITIS AND THE RHEUMATIC DISEASES

By Joseph L. Hollander, M.D.

The term "rheumatism" is derived from the Greek word *rheumatismos*,

Arthritis is the general term used when the joints themselves are the major seat of the Rheumatic Disease. *Fibrositis* denotes inflammation of connective tissue in any location, particularly around joints, and in or near muscles and tendons. *Rheumatology* is the study of the Rheumatic Diseases, including arthritis, rheumatic fever, fibrositis, neuralgia, myositis, bursitis, gout, and other conditions producing somatic pain, stiffness and soreness.

Arthritis is one of the oldest known, yet most neglected diseases. The earliest known example of multiple arthritis in a fossil vertebrate is in a [illegible]

tions).

Baillou (1538–1616) was the first to use the term rheumatism to designate a form of acute arthritis (as distinguished from gout); he also was the first to regard rheumatism as a clinical entity.

THE CONTRIBUTING EDITORS

NATHAN R. ABRAMS, B.S., M D , Cincinnati, Ohio

Assistant Clinical Professor of Medicine, University of Cincinnati College of Medicine. Director, Arthritis Clinic, Cincinnati General Hospital. Senior Attending Physician, Jewish Hospital Consultant in Internal Medicine (Arthritis), Veterans Administration.

VICTOR G BALBONI, A.B , M D , Boston, Massachusetts

Assistant in Medicine, Harvard Medical School and Massachusetts General Hospital. Visiting Physician, Robert Breck Brigham Hospital Consultant in Arthritis, Veterans Administration Hospital

EDWARD W. BOLAND, B S , M D , M S (Med), F.A.C.P., Los Angeles, California

Senior Staff Physician, St Vincent's Hospital

RALPH H BOOTS, B.S., M.D , F A C P., New York, N Y.

Director, Edward Daniel Faulkner Arthritis Clinic, Presbyterian Hospital Associate Clinical Professor of Medicine, College of Physicians and Surgeons, Columbia University Associate Attending Physician, Presbyterian Hospital.

MORRIS A. BOWIE, A.B., M D , Swarthmore, Pennsylvania.

Associate in Medicine, School of Medicine, University of Pennsylvania Assistant Professor of Physical Medicine, Graduate School of Medicine, University of Pennsylvania Director, Arthritis Clinic, and Attending Physician, Bryn Mawr Hospital Member, Council on Physical Medicine and Rehabilitation, American Medical Association.

HOWARD C. COGGESHALL, A B , M D , F A.C P., Dallas, Texas.

Associate Professor of Clinical Medicine, Southwestern Medical School, University of Texas Consultant in Medicine and Rheumatology, Dallas and McKinney V A Hospitals Consultant in Medicine to Office of Surgeon General, U S. Army, for Army and Navy General, and Brooke General Hospitals. Attending Physician, Baylor University Hospital, and Parkland City-County Hospital

RICHARD H FREYBERG, A.B., M S., M D , F A C P., New York, N. Y

Associate Professor of Clinical Medicine, Cornell University Medical College. Director, Department of Internal Medicine and Pediatrics, Hospital for Special Surgery. Director of Arthritis Clinics—New York Hospital and Hospital for Special Surgery Assistant Attending Physician, New York Hospital.

WALLACE GRAHAM, M.D., M R C.P (Lon), F R.C.P. (C), Toronto, Ontario.

Assistant Professor of Medicine, Faculty of Medicine, University of Toronto.

CHAPTER PAGE

Less Common Causes of Painful Shoulder 971
The Shoulder-Hand Syndrome . . . 971

55 DUPUYTREN'S CONTRACTURE (M A. BOWIE) 977

56 PAINFUL FEET (J G KUHNS AND T A. POTTER) 983
Normal Physiology of the Feet 984
Flat (weak) Feet 989
Proper Shoes 998
Common Abnormalities of the Feet . . . 1009
Immersion Foot—Trench Foot . 1013
Inflammatory Lesions of the Foot . 1015
Arthritis Involving the Feet . 1018
Other Abnormalities of the Foot . 1019

57. L[illegible] (P. D. W[illegible] AND P. D WILSON, JR.) 1023
[illegible] 1024
[illegible] 1031
[illegible] 1046
[illegible] 1059
[illegible] 1067

W. PAUL HOLBROOK, M D , F.A C.P., Tucson, Arizona.
Director of Research, Southwestern Clinic and Research Institute Senior Consultant in Medicine, St. Mary's Hospital. Senior Consultant in Medicine, Tucson Medical Center. Consultant in Medicine to the Surgeon General, USAF.

JOSEPH L. HOLLANDER, A B , M D , F A C P , Philadelphia, Pennsylvania
Associate Professor of Medicine, and Chief of Division of Rheumatology, Graduate School of Medicine, University of Pennsylvania Associate Professor of Clinical Medicine, School of Medicine, University of Pennsylvania Chief of Arthritis Section, University Hospital, and Chief of Rheumatology Department, Graduate Hospital, University of Pennsylvania Consultant in Rheumatology, Children's Hospital

JOHN G KUHNS, A B , M D., Sc D , F A C S , Boston, Massachusetts
Lecturer in Orthopedic Surgery, Boston University School of Medicine. Associate in Anatomy, and Instructor in Orthopedic Surgery, Harvard Medical School Chief of Orthopedic Service, Robert Breck Brigham Hospital. Visiting Orthopedic Surgeon, Boston Children's Hospital.

JOHN LANSBURY, M.D , C M., M S (Med), F A C P , Philadelphia, Pennsylvania.
Clinical Professor of Medicine, Temple University School of Medicine Chief of Arthritis Clinic, Temple University Hospital.

THEODORE A. POTTER, M D., Boston, Massachusetts.
Instructor in Orthopedic Surgery, Boston University School of Medicine Visiting Orthopedic Surgeon, Massachusetts Memorial Hospital Visiting Orthopedic Surgeon, Robert Breck Brigham Hospital.

CHARLES RAGAN, M D., New York, N. Y.
Associate Professor of Medicine, College of Physicians and Surgeons, Columbia University. Chief of Edward Daniel Faulkner Arthritis Clinic, Presbyterian Hospital Associate Attending Physician, Presbyterian Hospital Consultant in Medicine, Columbia Research Division, Goldwater Memorial Hospital

EDWARD F ROSENBERG, B A., M.D , M S (Int Med), Ph D (Int Med), F A C P , Chicago, Illinois
Chief of Arthritis Clinic, Michael Reese Hospital Member, Department of Medicine, Michael Reese Hospital

CHARLEY J. SMYTH, A B , M S , M.D., Denver, Colorado
Director of Graduate and Postgraduate Medical Education, and Associate Professor of Medicine, University of Colorado School of Medicine

PHILIP D. WILSON, A B , M D , F A C S , New York, N Y.
Clinical Professor of Surgery (Orthopedics), Cornell University Medical College *Surgeon-in-chief, Hospital for Special Surgery Attending Orthopedic* Surgeon, New York Hospital

PHILIP D. WILSON, Jr , M.D., New York, N. Y
Assistant in Orthopedic Surgery, Cornell University Medical College. Orthopedic Surgeon to Out Patient Department, Hospital for Special Surgery Surgeon to Out Patients (Orthopedics), New York Hospital

Classification of Arthritis (*Continued*)

Tabetic arthropathy of knee
Arthritis due to gout *Specify* joint. Record gout as primary diagnosis invariably.
New growths of joints *Specify joint and neoplasm,* as:
Synovioma of knee
Degenerative joint disease, multiple due to unknown cause; synonyms: osteo-arthritis (hypertrophic arthritis, degenerative arthritis, chronic senescent arthritis)
Hydrarthrosis, intermittent, of *Specify joint*
Periarticular fibrositis. *Specify joint*

Diseases in which arthritis, arthropathy or arthralgia is frequently associated (diagnose disease, list joint manifestation as symptom):

Acromegaly	Osteochondritis dissecans
Acute disseminated lupus erythematosus	Osteochondromatosis
Cyst of meniscus of knee	Periarteritis nodosa
Dermatomyositis	Psoriasis
Drug intoxication *Specify drug*	Pulmonary osteo-arthropathy
Erythema multiforme exudativum	Raynaud's disease
Erythema nodosum	Reiter's syndrome
Hemophilia	Scleroderma
Hysteria	Serum sickness
Ochronosis	Purpura, various types

Other Disturbances of Joints

I Disturbances of the joints secondary to abnormal postural strain
II Disturbances of the joints secondary to lesions of bone
III Disturbances of the joints associated with loose bodies
IV Disturbances of the joints secondary to functional or psychogenic causes (Psychogenic Rheumatism)

In the New York gr[illegible] and [illegible] for [illegible] f all types of arthritis is known, no grouping can be accurate The present classifications are based on clinical patterns, pathological changes in the tissues, and etiology when known

THE "SPECTRUM" OF RHEUMATIC DISEASES

Classification in joint disease can never be complete. Like the colors in the spectrum we may name and identify a certain number of types which are sufficiently different in our view to be separated and labeled with a name A rough separation of types of arthritis into groups is like identifying the primary colors Subdivision of the main groups into diseases is merely like identifying certain colors In the spectrum there are all shades between the named colors, and in the rheumatic diseases there are all variations between the identified types Variations occur in severity much like variations in intensity of specific colors, and mixtures of types, like

2

CLASSIFICATION OF ARTHRITIS

There are many different forms of joint disease, some caused by known etiologic agents, others from unknown etiologic factors. Some types are characterized by certain pathological and clinical features which makes their differentiation relatively easy. In other forms of joint disease the clinical pattern varies considerably, and identification may depend on detailed study. There are also numerous terms for certain types which are confusing. A few types of arthritis even remain *unproved as definite* clinical entities.

It is impossible to divide arthritis into acute and chronic forms, as almost any acute arthritis can pass into a subacute or chronic stage, and many cases [illegible] xacerbations. [illegible] (1) the frankly [illegible] cases that are possibly infectious but of unproved etiology; (3) cases representing degenerative forms of joint disease, which in Europe are termed arthroses; (4) cases in which the arthritis results from direct trauma to the joint; (5) ca [illegible] sa, [illegible] cl [illegible] Am [illegible] clature of Disease" should be used[6] The New York Rheumatism Association recently proposed another grouping of the forms of arthritis, using the same general terminology[5] (See Page 18).

Classification of Arthritis[11]

Arthritis due to infection. *Specify joint and organism when known*, as:
- Of spine due to tuberculosis
- Of knee due to gonococcic infection.
- Of wrist due to pneumococcic infection.
- Of hip due to syphilis

Arthritis due to rheumatic fever. *Record* rheumatic fever *as primary diagnosis invariably*

Arthritis, rheumatoid, of *Specify* as
- Arthritis, rheumatoid, multiple (synonyms: proliferative arthritis, atrophic arthritis, chronic infectious arthritis, Still's disease)
- Arthritis, rheumatoid, of spine (synonyms rheumatoid spondylitis, atrophic spondylitis, Marie-Strumpell disease, spondylose rhizomelique, infectious spondylitis, spondylitis ankylopoietica, von Bechterew's disease).

Arthritis due to direct trauma of . *Specify joint and trauma*, as:
- Of knee due to contusion
- Of elbow due to habitual dislocation
- (This includes traumatic forms of synovitis and degenerative arthritis secondary to old trauma, sprains, strains, internal derangements, fractures into joints, loose bodies and damage to cartilages, etc)

Neurogenic arthropathy (Charcot joint) (associated with tabes dorsalis, syringomyelia, cord trauma, nerve injuries or leprosy). *Specify joint*, as:

used alone as a diagnosis, tells nothing because we must know which "colors" are mixed, and in what proportion. In a case where rheumatoid

THE PREVALENCE AND PROBLEM OF RHEUMATIC DISEASE

Rheumatic diseases are the commonest cause of chronic illness in this country and rank second in causing temporary and permanent disability.[18] It has been estimated that over 10,000,000 people (at least 1 in every 20 persons) in the United States have some form of rheumatic disease [21] Rheumatism is more common than the total number of cases of tuberculosis, diabetes, cancer and heart disease combined [19]

Arthritis surpasses injury from accidents from the standpoint of days lost from work. The rheumatic diseases disable ten times as many persons per year as does diabetes or tuberculosis and seven times as many as cancer As a cause of days lost from work from chronic disorders in this country

kil

should be put at the head of all other chronic diseases as of preeminent medical, economic and social importance Unfortunately, a large percentage of individuals affected by the rheumatic diseases receive no medical treatment or treat themselves [21]

anc

chr

insured population [8]

It has been estimated that on a given day in this country about 1 person in each 1000 develops a rheumatic disease [20] More than 320,000 otherwise able individuals are rendered unemployable in the United States for an entire year by rheumatic diseases National Health Surveys have shown that the rheumatic diseases were the sixth greatest cause of disability and

times as common among individuals on relief as in the higher income group [12]

Of

actua

anoth

2. *Joint swelling* or enlargement, which may be one or more of four types
   A. *Periarticular edema*, a *diffuse* edema of subcutaneous tissue about a joint, extending beyond the reflections of the synovium, or *localized* in adjacent bursae or tendon sheaths
   B *Synovial or capsular thickening*, recognized as a doughy enlargement confined to the areas where the joint capsule and synovial reflections are near the surface.
   C. *Effusion*, or hydrops, which is identified by the demonstration of fluctuation in the joint sac, or by bulging limited to the reflections of the synovium. (In the knee, effusion is readily demonstrable by ballottement of the "floating patella" against the femur) Bursal effusion, as in "housemaid's knee," is extra-articular.
   D. *Bony enlargement.—Osteophytes* of sufficient size to be palpable are commonly found at the articular margins in degenerative joint disease (osteoarthritis). Spur formation at the articular margins of the terminal phalangeal joints of the fingers accounts for much of the enlargement of Heberden's Nodes Gross distortion of the bone ends may be noted in acromegaly, or locally in osteogenic sarcoma or other bone tumors arising near a joint.

3 *Tenderness* may be *localized* (as at a point of injury), or *diffuse*, about the entire joint The *degree of tenderness* is important

4 *Crepitation on motion* —"Grating" or "grinding" on motion is common in degenerative joint disease Presence of this finding usually indicates a roughening of the opposing cartilages, which may be from age changes, chondromalacia, injuries, abnormal strains, previous inflammatory changes, osteochronditis, or other conditions which have caused irregularities in the surface of the articular carilage This must be differentiated as
   A. *Fine "rubbing" crepitation* felt over rheumatoid arthritic joints from presence of granulation tissue (pannus), or from bone ends denuded of cartilage by pannus
   B. *Coarse crepitation* or "crackling" so commonly felt in the knees [illegible]

   [illegible] demonstrable joint disease (Generally, the coarser the crepita- [illegible]

5 [illegible] or injury, producing actual *disloc*[illegible] bones, with demonstrable malalignment.

6 *Limitation of motion* of the joint The range of joint motion may be demonstrably decreased by·
   A *Swelling* of a degree sufficient to *distend* the *capsule* tightly
   B *Inflammation* of the *synovium* severe enough to cause reflex sustained contraction (*spasm*) of the muscles that move the joint (*guarding*).
   C *Fibrosis or contracture of* the synovium or *capsule*
   D *Shortening of muscles and tendons* which move the joint (*contracture*).
   E *Fibrous or bony ankylosis*, caused by adhesions across the joint space or bony bridging with complete fixation of the join[illegible]

Classification of Arthritis prepared by the New York Rheumatism Association[6]

A Infectional arthritis of proved etiology
B. Possibly infectional, etiology unproved
  1. *Arthritis of rheumatic fever*
  2 Rheumatoid arthritis (atrophic, chronic infectious)
    (a) Adult type
    (b) Juvenile type (Still's disease)
    (c) Ankylosing spondylitis (Marie-Strumpell, etc)
    (d) Psoriatic arthritis
  3 Arthritis associated with various infections
C Degenerative joint disease
  1 Osteoarthritis (hypertrophic, degenerative)
    (a) Generalized
    (b) Localized
      (1) Secondary to previous trauma
      (2) *Secondary to structural abnormality*
      (3) Secondary to previous infectional arthritis
      (4) Cause unknown
D Arthritis associated with disturbance of metabolism
  1 Gout
  2 Others
E Arthritis of neuropathic origin
  1 Secondary to tabes dorsalis
  2 Secondary to syringomyelia
F Neoplasms of joints cyst, xanthoma, hemangioma, giant-celled tumors, synovioma
G Mechanical derangements of joints
  1. Traumatic arthritis
  2 Joint disturbance secondary to abnormal postural strain
H. Miscellaneous forms
  1 Manifestations of systemic disease
    (a) Arthritis of serum sickness
    (b) Arthritis of hemophilia
    (c) Intermittent hydrarthrosis
    (d) *Pulmonary osteo-arthropathy*
    (e) Hysterical joints
  2 Local joint disturbances
    (a) Aseptic bone necrosis
      (1) Secondary to contusion, fracture, dislocation or air embolism
      (2) Of unknown etiology (juvenile osteochondritis, Legg-Calvé-Perthes' disease, Kohler's disease, Freiberg's disease Osgood-Schlatter's disease)
    (b) Osteochondritis dissecans
    (c) Osteochondromatosis

mixtures of known colors, may produce a different "color" or clinical pic-
... forms of arthritis is unknown, it might
s as we do of the spectral bands—a
her than as definite "pigeonholes"
Certainly rheumatoid arthritis and

PHYSICAL SIGNS OF JOINT DISEASE

The important physical signs of joint disease are:

1. Abnormalities of *color, consistency, temperature*, or amount of *sweating* of the *overlying skin*. Skin lesions or ulcers near the joints occur in some types.

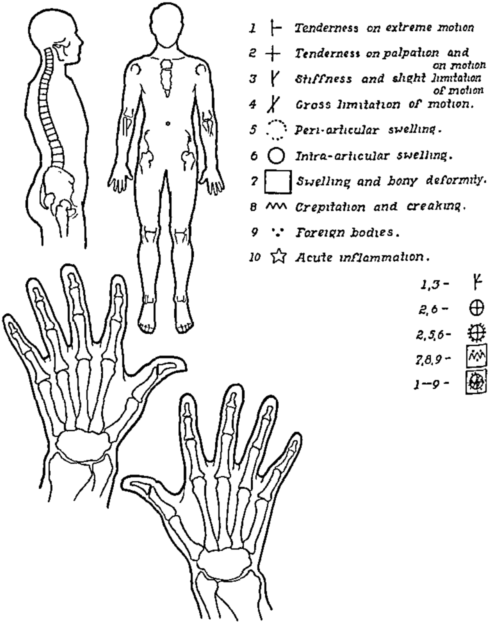


FIG 1 —Arthritic record chart, using Jansen ciphers.

The lack of facilities for proper care of arthritic patients in this country is appalling In a recent survey, it was shown that there were not more than a total of 200 free hospital beds in the entire country specifically available for arthritic patients as compared with more than 100,000 free beds for the care of tuberculous patients.[17] Furthermore, although $100,000,000 is available for research work in tuberculosis, less than $10,000,000 is available for similar projects in arthritis These facts must be emphasized to the general public so that adequate facilities and money is made available at once for the proper care and study of arthritic patients.

The Arthritis and Rheumatism Foundation, established in 1948 under the sponsorship of the American Rheumatism Association, has undertaken the task of conducting an annual nation-wide campaign to raise funds, which are dispensed and administered for:

A Development of a nationwide *research program* to discover the cause, and improve the methods for the treatment, cure, and prevention of arthritis and other rheumatic diseases

B Establishment of *fellowships* to increase the number of men and women qualified to conduct research and specialize in the proper diagnosis, treatment, and prevention of rheumatic diseases, and also teach other physicians adequately in this field

C. Development of *centers* devoted to research, teaching and treatment in arthritis and allied conditions

D Promotion of better *medical* and *public understanding* of the extent of social and human costs imposed by the rheumatic diseases, and of what can be done to assist in their alleviation, control and prevention.

During the past four years the Arthritis and Rheumatism Foundation has made great strides toward the goals outlined above. The first great achievement was the sponsoring of the Seventh International Rheumatism Congress in New York in 1949 Since this time, the money raised in the

explaining the types of arthritis, the treatment for them, and the problems still to be worked out This aid to physicians in the fight against this

largely at the instigation of the Arthritis and Rheumatism Foundation

RELATIVE INCIDENCE OF THE RHEUMATIC DISEASES

In civilian populations many attempts have been made to determine the relative frequency of the various types of arthritis and related conditions The difficulties are great. Actually about 97 per cent of all persons who live beyond middle life have some rheumatic complaints—mostly due to degenerative joint disease.[11] The complaints usually are not severe enough, nor the duration of painful periods long enough to force many of these individuals to seek medical attention, but degenerative joint disease

excessive sweating, *nodules* (noting location, size, consistency, and fixation to underlying tissues), edema, or hardness of tissues between joints (as in scleroderma).

3 *Move joints to determine: range of motion*, existence of *contractures, subluxations, pain* on *motion, preternatural mobility* (from injury—torn capsule or ununited fractures near joints-Fig 5), *fibrous ankylosis* (slight motion possible, usually), or *bony ankylosis* (complete fixation)

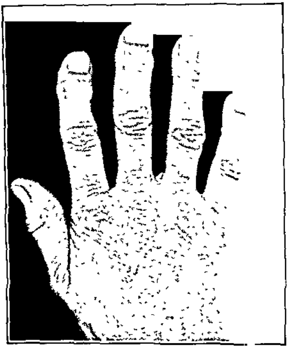


FIG 3 —Degenerative joint disease of the hand Heberden's nodes seen at the distal interphalangeal joints (in a woman aged fifty-eight years)

4 *Have patient move joints to determine. strength of grip, range of active* ...ents

4)
...ith
...y in

symmetrical joints on the two hands, swelling is spindle shaped (PIP joints), boggy, and tender Joints are painful on motion, restriction of motion particularly extension) is common, and *atrophy of the intrinsic muscles* of the hands, and of the forearm muscles is usual (weak grip). (Fig 2)

ESTIMATED ANNUAL NUMBER OF DAYS LOST FROM WORK OR OTHER USUAL PURSUITS BY REASON OF SPECIFIC CHRONIC DISEASES (UNITED STATES, 1937)

Disease	*Annual Number of Days Lost*
Nervous and mental disease	132,500,000
Rheumatism	97,200,000
Heart disease	95,200,000
Arteriosclerosis and high blood pressure	56,900,000
Tuberculosis, all forms	41,400,000
Cancer and other tumors	36,300,000
Nephritis and other kidney diseases	28,400,000
Diseases of the female organs	26,800,000
Hay fever and asthma	21,900,000
Diseases of gallbladder and liver	20,000,000
Diabetes mellitus	19,200,000
Ulcers of the stomach and duodenum	13,600,000
Hernia	13,600,000
Chronic diseases of the skin	10,300,000
Anemia	8,500,000
Diseases of the bladder and urethra	8,200,000
Chronic bronchitis	8,000,000
Chronic appendicitis	7,600,000
Goiter and other thyroid diseases	7,600,000
Other diseases of the circulatory system	6,600,000
Sinusitis	6,000,000
Varicose veins	5,900,000
Chronic tonsillitis and other throat infections	5,400,000
Hemorrhoids	5,100,000
Chronic pleurisy	4,200,000
Chronic diseases of the eye	4,200,000
Chronic diseases of the ear	3,300,000
Diseases of the prostate and male genito-urinary organs	3,300,000

From Preliminary Reports, the National Health Survey, Sickness and Medical Care Series Bulletin No 6, U S Public Health Service, Washington, D. C., 1938 [19]

work each year. Over 50 per cent of the disability is in persons under forty-five years of age [11]

The recent U S. Public Health Survey,[21] carried out in late 1951, shows

change of type of work was necessary because of rheumatic disease in 1% of the *entire population* by age 40, 2% by age 50, and 4% gy age 60 That this is an important factor in labor turnover is obvious.

The importance of the problem of the rheumatic diseases to Industry is ... The Government and various other social ... dous disability caused by these

... nd *problems of caring for the* victims of arthritis is now being undertaken by the National Institute of Arthritis and Metabolic Diseases, United States Public Health Service. The survey was originally instigated by the Arthritis and Rheumatism Foundation. The need for proper and adequate hospital facilities, and for home care plans will be determined, so that an organized program of arthritis treatment can be made available throughout the country.[4]

7. *Muscle changes.*—*Weakness,* hyperactive stretch reflex (*spasm*), *atrophy,* and *shortening* are usually more prominent in the flexor muscles of a diseased joint, and produce marked deformity or limitation of motion. These changes are most marked in rheumatoid arthritis, and are possibly caused by inflammatory changes in the muscles and nerve sheaths (see Pathology of Rheumatoid Arthritis).



Fig. 2.—Rheumatoid arthritis. Note the fusiform swellings and deformities in the proximal interphalangeal joints of this young woman.

8. *Nodules.*—*Rheumatoid nodules* are frequently found adjacent to finger, elbow or knee joints, as are the *tophi* of gout. Small *loose bodies* or "joint mice" can occasionally be felt in the knee—these can be moved under [illegible] disappear when palpated. Nodules or tophi near [illegible] psule, and are not [illegible] often found at the [illegible]

TECHNIQUE OF JOINT EXAMINATION

The technique of examination of joints is given in detail for the specific joint, or group of joints to be studied.

Examination of Fingers and Hands.—Record positive findings. 1. *Look for: distribution of joint involvement* (symmetrical or asymmetrical between left and right), *joints involved*—(Figs. 2, 3 and 4)— terminal interphalangeal (TIP), proximal interphalangeal (PIP), or metacarpo-phalangeal (MCP), with number of finger (thumb-1, little finger-5), *type* and *degree of swelling*

As a result of army experience in World War I, it was estimated that the order of frequency in occurrence of the rheumatic diseases in World War II would be fibrositis (muscular rheumatism)—most common; then, in order, rheumatic fever, rheumatoid arthritis, septic arthritis, degenerative joint [illegible]

Three were for treatment of rheumatic fever, and two for various types of arthritis and allied conditions

Principal Diagnosis in over 5200 Patients Admitted to the Army Rheumatic Disease Center at Ashburn General Hospital[9]		
Rheumatoid Arthritis	1840 cases	35%
[illegible]	988 cases	
	256 cases	
	499 cases	
	97 cases	
	1449 cases	28%
Involving spine	955 cases	
Involving knees	404 cases	
Involving feet	325 cases	
Involving hips	83 cases	
Involving fingers	43 cases	
Involving other joints (shoulders, elbows, etc)	42 cases	
Psychogenic Rheumatism (psychoneurosis with musculo-skeletal reference of symptoms)	893 cases	17%
Fibrositis (including bursitis, lumbago, etc)	151 cases	3%
Specific Infectious Arthritis	115 cases	2%
Gonococcal arthritis (proved)	41 cases	
Reiter's Syndrome	57 cases	
Tuberculous Arthritis	4 cases	
Septic Arthritis (Staph or Strept)	8 cases	
Miscellaneous infectious types	5 cases	
Orthopedic Conditions	644 cases	12%
Spinal Deformities and Backstrain	362 cases	
Footstrain or Deformity	119 cases	
Ruptured Intervertebral Disc	89 cases	
Internal Derangement of Joint	51 cases	
Miscellaneous Deformities, tumors, etc	23 cases	
Rheumatic Fever*	36 cases	0 7%
Gout	21 cases	0 4%
Miscellaneous and Undiagnosed	72 cases	1 4%

* *Soldiers with rheumatic fever were usually sent to other hospitals, so the* relative incidence was very low

Complete figures on the incidence of rheumatic fever during the War are not yet available, but from 1942 to 1944 inclusive nearly 15,000 cases had been reported in the United States Army[7] (See also Chapter 35).

skin lesions, nail changes, changes in *color,* or *atrophy of skin, muscle atrophy, excessive sweating, deformities,* or *shortened fingers*

2. *Palpate for: degree of tenderness, type of swelling,* heat or coldness, excessive sweating, *nodules* (noting location, size, consistency, and fixation to underlying tissues), edema, or hardness of tissues between joints (as in scleroderma)

3. *Move joints to determine: range of motion,* existence of *contractures, subluxations, pain* on *motion, preternatural mobility* (from injury—torn capsule or ununited fractures near joints-Fig. 5), *fibrous ankylosis* (slight motion possible, usually), or *bony ankylosis* (complete fixation).



FIG. 3 —Degenerative joint disease of the hand Heberden's nodes seen at the distal interphalangeal joints (in a woman aged fifty-eight years)

4 *Have patient move joints to determine· strength of grip, range of active motion,* muscle *weakness* or atrophy, or loss of certain active movements [illegible]

particularly extension) is common, and atrophy of the intrinsic muscles of the hands, and of the forearm muscles is usual (weak grip). (Fig. 2)

(osteoarthritis) is the most common form of rheumatic disease observed in several large arthritis clinics.[9]

Another common form of rheumatic disease is fibrositis. Nearly everyone

would not reflect the true incidence of such a disorder.

The most common of the more severe forms of rheumatic disease are *rheumatoid arthritis* and *rheumatic fever*. The latter is the chief cause of death from disease in children aged five through nineteen,[6] and produces more chronic heart disease than all other causes combined. Rheumatoid arthritis, though it most commonly attacks later (twenty to fifty years of

actually number only 27 per cent of the total patients registered in the Arthritis Clinic of the Hospital of the University of Pennsylvania,[9] 82 per cent of the revisits to the clinic are by this group.

and begin to look forward with dread to crippling deformities and a wheelchair existence. It is because of the dread that "arthritis" conjures up in the minds of most, that physicians should know how to diagnose correctly the forms of rheumatic disease *Reassurance, to a patient who has a milder form of arthritis than the rheumatoid type, may be the best treatment the physician can give.* For this reason also, the term degenerative joint disease is better than osteoarthritis.

THE RHEUMATIC DISEASES AS A MILITARY PROBLEM

Among the 4,000,000 soldiers in the American Army of the first World War there were reported 33,613 instances of rheumatoid arthritis and

connected rheumatic disease, and these men receive an annual pension which totals over $10,000,000.[11]

In the British Expeditionary Forces (1940), 26 per cent of all medical patients admitted to several General Hospitals had rheumatic disorders, the most common of which was fibrositis (70 per cent), with 15 per cent

dence of rheumatoid arthritis include emotional disturbances, exposure to dampness, prolonged chilling, exposure to respiratory infections, etc.[9]

(Fig. 6) is frequent, marked muscle atrophy is seen, and ankylosis in deformed positions is frequent

In *degenerative joint disease* bony enlargement of the terminal interphalangeal joints (Heberden's nodes) is characteristic (Fig. 3), with limited motion but no ankylosis or muscle atrophy. Some deviation deformity of the TIP joints may be seen. Both hands are usually involved. Proximal interphalangeal joints (PIP) may become involved with a similar enlargement (Bouchard's Nodes) years after development of nodes on TIP joints

In *gouty arthritis* the distribution of involved joints is asymmetrical, and nodules in or about the joints produce irregular swellings. Tophi may ulcerate, and large tophi can completely destroy a joint or cause spontane-

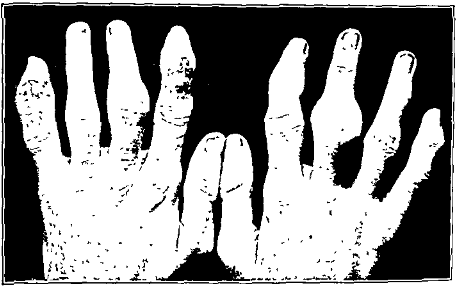


FIG 4 —Gout Note asymmetrical swellings and drainage from ulcerated tophi

ous amputation of a finger, late in the disease Any joints may be involved. Gouty involvement of the hands is uncommon. (Fig. 4.)

In *causalgic states* (as in the "shoulder-hand" syndrome) the hand may be diffusely swollen, tender, discolored, wet, and painful on motion. Flexion contractures of fingers (*e. g.*, Dupuytren's contracture) are common. The skin may be tense and shiny.

In *scleroderma* the skin is tense, shiny, and atrophic. Subcutaneous tissues at first are tensely swollen, later shrunken and fibrotic. Joints may be tender and enlarged. Shortening of the distal phalanges may be seen in late cases. Flexion contractures of the fingers are usual.

In *gonococcal arthritis* only one or two finger joints (if any) will be involved. Tenosynovitis, with marked periarticular edema often is noted over the dorsum of the hand when the wrist is involved.

CHAPTER 2

EXAMINATION OF THE ARTHRITIC PATIENT

By Joseph L. Hollander, M.D.

Successful management of joint disease can come only after accurate diagnosis. *A complete study of the patient with joint complaints is essential even though the diagnosis may seem obvious.* The history of the case must cover the pertinent facts. The physical examination must cover not only the general disorders looked for in all patients, but a *careful and complete examination of the joints must be made.* Although any well trained interne can make a competent physical examination of the heart, lungs, or abdomen, *the physician is rare indeed who knows how to examine joints.* In the

HISTORY IN JOINT DISEASE

1. After the *age, sex,* and occupation of the patient are noted, the arthritic history may well begin with the *family history* of rheumatic disease. There is a hereditary tendency in many forms of arthritis, notably rheumatoid arthritis, rheumatic fever, degenerative joint disease (particularly of hands), rheumatoid spondylitis and gout. Although most patients know of no *specific* joint disease in the family, in those cases where the history reveals a family incidence a valuable clue may be obtained.

2. *The past history* is often significant. The recent or remote incidence of

recurrent for many years. A fair proportion of patients with rheumatoid arthritis have a past history of rheumatic fever or of frequent sore throats. "Post-gonorrheal rheumatoid arthritis" was frequently encountered in the Armed Forces during World War II. Weakness or deformities from polio-

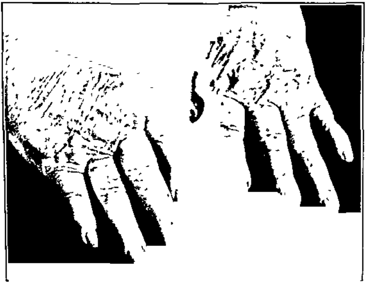
development of many joint complaints in later years. History of previous allergies, endocrine disorders, or other *systemic diseases* may be significant

(The characteristic hand deformities of leprosy, pulmonary osteo-arthropathy etc. are described in the chapter on Differential Diagnosis of Rheumatoid Arthritis p. 215.)

Examination of the Wrists.—The wrist is a compound joint—a *group* of joints *Inspection* quickly reveals the presence or absence of swelling or deformity Predominence of flexor muscle strength in severe arthritis of



FIG 5 —Juvenile Rheumatoid Arthritis, duration ten years, in a girl of sixteen Ankylosed finger joints had been "mobilized" by manipulation performed by patient's mother several years before photograph was taken Fractures of phalanges near ankylosed joints have produced multiple "flail joints" Wrists and elbows are ankylosed



Over 14,000 cases of arthritis and related diseases were treated at the Army Rheumatism Centers at Army and Navy General Hospital, Hot Springs, Arkansas, and Ashburn General Hospital, McKinney, Texas.[9]

The relative incidence of the rheumatic diseases among soldiers was not as anticipated, in some instances because the advent of sulfonamides and penicillin had reduced septic arthritis (particularly gonococcal) to a very low rate of occurrence, and because better understanding of the difference [illegible]

The methods of group treatment in teaching large numbers of arthritis patients how to "live with the disease" and how to "help themselves" used at the Army Arthritis Centers should establish a pattern for civilian clinics. The Reconditioning Programs emphasized proper physical therapy, exercise, and mental reorientation for all the soldiers with rheumatic disease. This emphasis on convalescent care and training also has been carried over into civilian medical practice to the great benefit of arthritic patients

The physicians who served at the arthritis centers learned a great deal about the diagnosis and management of the rheumatic diseases The patients with arthritis were better cared for because of this experience, and more comprehensive and accurate clinical observations were made possible.[7]

BIBLIOGRAPHY

1 Britten, R H Pub Health Rep, 55, 2199, 1940
2 Britten, R H, Collins, S D and Fitzgerald, J. S Pub Heath Rep 55, 444, 1940
3 Editorial Lancet 1, 145, 1943
4 Hartung, L L, et al Arthritis Clinic Manual, Arthritis and Rheumatism Foundation, New York, 1952
5 Hench, P S Chronic Arthritis, in Barr's Modern Medical Therapy in General Practice, Baltimore, Williams and Wilkins Co, 3, 3298, 1940
6 Hench, P S et al Ninth Rheumatism Review Ann Int Med 28, 66, 1948
7 Hench, P S and Boland, E W Ann Int. Med 26, 808, 1946
8 Hench, P S, Osgood, R B, and Wainwright, C W Army Med. Bull. 60, 1942
9 Hollander, J L Unpublished Data
10 Horsmell, R S New England Jour Med, 223, 754, 1940
11 McEwen, Currier, et al Primer on Rheumatic Diseases, Jour. Am Med Assn, 139, 1068, 1949
12 Murphy, G E Bull Hist Med, 14, 123, 1943
13 Osgood, R B Am Jour Med Sci, 200, 429, 1940
14 Pemberton, R, and Scull, C. W Med Clin, North America 24, 1591, 1940
15 Pepper, O H P War Med 1, 463, 1941
16 Ragan, C and Meyer, K Ann Rheum Dis, 7, 35, 1948.
17 Snyder, R G Ann Int Med, 13, 1317, 1940
18. T [illegible]
19. T [illegible]
20 T [illegible]
21 Woolsey, T D Public Health Reports, 67, No 6, 505, 1952

the wrist usually produces deformity in palmar flexion and ulnar deviation (note left wrist in Figure 5). *Palpation* reveals the type of swelling, degree of tenderness, and particularly localized swellings, such as ganglia (synovial cysts) *Motion* must be tested in *dorsiflexion* (normally 70°, usually the first movement restricted), *palmar flexion* (normally 80°), and *radial* and *ulnar deviation* (normally a total range of 60°). Comparison with the opposite wrist, if not also involved, or estimation by comparison with the examiner's own movements may be satisfactory enough for ordinary clinical record, but, for later reference, measurements made with a *goniometer* (an instrument for measuring angles) are much more accurate in noting actual degrees of motion. *Weakness* and *atrophy* of *forearm muscles* should be noted. Measurements of circumference of the forearm at a given distance below the elbow give a valuable record of degree of atrophy. Infectious arthritis, such as the gonococcal and tuberculous forms, may involve *one* wrist, but rheumatoid arthritis often will involve *both*. Localized pain and tenderness at the radial styloid indicates stenosing tenosynovitis (DeQuervain's disease). This latter condition may be further diagnosed by the "Finklestein test," in which the thumb of the patient is held in his clenched fist and the examiner forces the wrist into ulnar deviation. This motion produces acute pain if DeQuervain's disease is present.

Examination of the Elbow.—The elbow joint has two distinct functions. The humero-ulnar joint is a hinge joint permitting *flexion* and *extension*. The humero-radial joint permits rotation of the head of the radius, making possible *supination* and *pronation* of the forearm. Mild to moderate degrees of *swelling* and *tenderness* are most noticeable on the extensor surface, just medial and lateral to the olecranon *Nodules* should be looked for *in the olecranon bursa* and *along the ulna* distal to the olecranon. *Effusion* into the *olecranon bursa* is frequently found after *trauma* and in *gout*. The first nodules in a patient with rheumatoid arthritis are nearly always found near the elbow. *Motion* should be evaluated with a goniometer, recording limitations of *flexion* or *extension* in degrees (normal range 45° to 180°). Estimation of the range of *supination* and *pronation* (normal about 140°) [illegible]

[illegible]

rheumatoid arthritis (Fig. 5). *Active motion* of the elbow reveals any weakness of the biceps, triceps, or brachialis muscles. Atrophy in rheumatoid arthritis is severe, and muscle tenderness is sometimes marked. The [illegible] should

[illegible] *apulo-humeral joint* is a ball and socket joint permitting motion in all directions, including rotation. Shoulder movement as we know it is more complex than simple scapulohumeral motion, however. Motion is assisted by *sliding* of the *scapula* over the posterior chest wall, with some movement of [illegible] *Inspection* of the shoulder [illegible] pment. In arthritis [illegible] or pectoral muscles may be observed *Palpation* will often confirm the presence of moderate

disease, the proximal interphalangeal joints in rheumatoid arthritis, or the sacroiliac joints in rheumatoid spondylitis). The *rate of spread* of the arthritis to new joints, and the *type* of *spread* are noteworthy (*e. g.*, migratory and transient, as in rheumatic fever, or slowly involving additional joints as in rheumatoid arthritis). *Symmetry of involvement* of joints is usual in rheumatoid arthritis (*e. g.*, both knees, both third proximal interphalangeal joints, both wrists), but less frequent in other forms of arthritis

8. *Joint swelling.*—This may be described as transient "puffiness" seen in the morning or evening, or as persistent, severe and painful swelling. *Localized swelling* of specific joints must be differentiated from a *diffuse periarticular edema* which involves a whole hand or foot The presence of *heat* and *skin discoloration* over an involved joint may be described by the patient, as well as the degree of *tenderness*.

9. *Systemic reaction.*—The patient should be questioned concerning the occurrence of *fever, chills, anorexia, weight loss, weakness, sweating,* cessation of or *changes* in the *menstrual cycle*, or *local infections* coexisting with or complicating the arthritis.

10 *Course* —This may be *steadily progressive, stationary*, or *recurrent* with exacerbations and partial remissions, acute exacerbations with complete remissions (as in early gout), rapidly progressive, etc

from these simple remedies) *Previous hospital care*, or *bed rest*, previous

worthy

12. *Present disabilities and deformities* —Joints particularly involved at the time of interview, and limitations imposed on the patient's normal activity by contractures, ankylosis, muscle weakness, or severe pain on use are important for later comparison.

GENERAL PHYSICAL EXAMINATION IN ARTHRITIS

The physician needs no reiteration of the procedure for general physical examination of a patient To be looked for particularly, in arthritic

3 *Subcutaneous nodules* are commonly found in some forms of arthritis (nodules in rheumatoid arthritis usually found on extensor surface of forearm in or below olecranon bursa, tophi of gout on ear margins, etc)

4 Possible *foci of infection*, such as carious teeth, infected tonsils, nasal obstruction, enlarged, tender prostate, or pelvic disorders should be noted for future treatment

5. *Cardiac abnormalities, lymphadenopathy, splenomegaly, hepatomegaly* and *muscle reflex changes* are sometimes produced by rheumatic diseases, and should be looked for particularly.

be elicited.
and tender-
joints and
acromioclavicular joints when present *Motion* will quickly reveal limitations in range in *abduction* (raising to the side), *anterior elevation* (or flexion forward), *posterior elevation* (extension backward), *external rotation* (flex elbow, rotate arm laterally), *internal rotation* (place arm across back-"reach for the hip pocket"). *Pain on motion*, and particularly *location* and *position* of arm when pain is noted, or visible *muscle spasm* or involuntary contraction produced by sudden pain, should be noted *Crepitation* is commonly noted on shoulder motion. This may be felt over the scapulo-humeral joint, over the sternoclavicular joint, or over the angle of the scapula (fibrous crepitation). *Abduction*, and *internal* and *external rotation* are usually all painful and limited with inflammation of the true shoulder (scapulo-humeral joint), or closely adjacent structures (as in periarthritis, subdeltoid bursitis, or supraspinatus tendonitis). (For further details, see chapter on The Painful Shoulder).

Examination of the Temporo-mandibular Joints.—Involvement of the articulation of the jaw is rather common in rheumatoid arthritis. On *palpation* both *tenderness* and *crepitation* on motion may be felt. *Motion* is easily tested by ... itation is usually recor... lower teeth with the ... may result from trauma, or may follow inflammatory change. The trauma may be minimal—such as strain from sleeping with a pillow pressing the jaw to one side Ankylosis of the temporo-mandibular joints is occasionally found

... *Palpation* will reveal local areas of *tenderness*, and perhaps *crepitation* on rotation when osteoarthritic changes are present. *Spasm* may be palpable, particularly over the trapezius muscle, or the scalenus anticus *Arthritic changes in the cervical spine* frequently account for *pain in the shoulder and arm* (brachial neuralgia)

chest ...

normal motion possible.

Examination of the Spine.—The details of this somewhat complicated examination are given in the chapters on Spondylitis and on Backache. A few general pointers will be given here

1. W...
dorsal
curve,

Previous surgical operations should be noted, particularly tonsillectomy, hysterectomy, lithotomy, or sinus operations. The development of degenerative joint disease (particularly Heberden's nodes) may follow surgical menopause even in a young woman. Gouty arthritis is often preceded by the development of renal calculi. Focal infection may be indicated by the history of previous sinus operations.

3. *Predisposing factors.—Emotional traumata* and periods of mental stress often precede the onset of a rheumatoid arthritis. History of *chilling, exposure,* or *abnormal working conditions* may precede joint symptoms. *Chronic fatigue* is frequently noted *Abnormal dietary habits,* either in deficient diets, or overindulgence in certain foods such as meat, or *abuse of alcohol* are important points to be noted. *Excessive use of tobacco,* and ingestion of certain *drugs,* such as sulfonamides, may provide a clue to the nature of the complaint in some cases. It is becoming increasingly apparent that prolonged or abnormal stresses are intimately associated with the onset of the rheumatic diseases.

4. *Prodromal symptoms.*—Increasing fatiguability, anorexia, weight loss, stiffness, and vague, transient aches and pains are usually the first signs of rheumatoid arthritis Attacks of gout are frequently ushered in by periods of excessive craving for food, restlessness and irritability. Mild recurrent, transient pains and stiffness after rest are usually the prodromes of degenerative joint disease

5. *Mode of onset* —This may be *acute,* with fever, rapid development of joint pain and swelling, etc., *subacute,* with little systemic reaction, but fairly rapid development of joint symptoms, or *insidious* with the gradual development of mild symptoms slowly increasing to the point of sufficient annoyance to the patient that he realizes something is wrong. Any *infectious process,* or other possible *precipitating factors* present at the time of onset should be noted The *date of onset* should be determined, as nearly as possible

[illegible]

and it may take hours for the patient to "limber up." In degenerative joint disease (osteoarthritis) the stiffness is usually most troublesome after periods of rest in one position (*articular jelling*), and disappears quickly on limbering up exercise or resumption of normal activity. "Jelling" is also noted in other types of arthritis The *effect of weather* on the joint symptoms is important Most patients with rheumatic disease of any type find the symptoms increase before *weather changes,* and do not vary with the type of weather alone The *effect of rest and exercise* on joint symptoms is noteworthy, as is *aching with fatigue,* and the time of day symptoms are most pronounced (*e g*, night pain in gout and degenerative joint disease, "vesperal aching" in rheumatoid arthritis, etc.). Pain from strain or deformities is more marked toward evening. The *amount and type of pain* should be recorded. The excruciating, burning, boring pain of gout is characteristic, and a steady, dull ache must be differentiated from sudden stabs of pain on attempted movement, etc.

7. *Distribution of Joint Involvement.*—The *location* of the original joint involved often gives a diagnostic clue (*e g*, the "bunion joint" in gout, the shoulder in bursitis, the distal phalangeal joints in degenerative joint

curvature, or *lordosis,* may be noted, or *flattening,* with the pelvis rotated forward and upward.

2. *With the patient standing, and viewing from behind, note presence of scoliosis,* or deviation from a straight line. *Tilting of the pelvis,* from short leg or flexion deformity of a knee or hip, will produce a lateral curvature of the spine to compensate for the imbalance. The paravertebral muscles (erector spinae) on the shortened side will appear more prominent. *Sharp angulation* may be noted from structural abnormality of the vertebrae. Measure *leg length* from anterior superior iliac spine to internal malleolus

3 *Motion* of the spine should be tested by having patient bend (*flexion*) [illegible]

Lateral deviation (bending to left and right) is principally a motion of the *lumbar vertebrae* when the patient *stands.* When *sitting,* the *dorsal,* or thoracic, vertebral joints are called into play. (Have patient sit on table, shoulders hunched forward. and elbows bent. Direct patient to touch point of elbow to table beside him without raising opposite buttock from table.) *Rotation* of the spine should be tested both sitting and lying supine In sitting, the patient is directed to turn to left and right (pelvis fixed), and limited motion or spasm thus elicited is noted. In lying supine the patient is directed to keep shoulders flat on table, while pelvis is rotated to [illegible]

extended).

5. *Measure chest expansion* —This is a composite motion of the costovertebral joints. (For other important tests for spinal disorders, see under chapter on Low Back Pain).

Examination of the Hips.—With the patient lying supine the legs normally lie flat with slight rotation of the pelvis producing some lumbar lordosis If the hips are slightly flexed, the lordosis flattens out. A quick test for hip function is the "heel to knee test " The hip and knee are flexed so that the heel lies beside the opposite knee, then the hip is externally rotated as if to place the lateral surface of the bent knee on the table. This movement combines *flexion, abduction,* and *external rotation. Pain, limitation,* and *spasm* of the *adductor muscles* indicate a hip disorder. *Flexion* of the hip is normally possible to about 45° with the spine. *Abduction* is normally possible to 140° with the spine (neutral position is with the femur at 180° with the spine), *adduction* (crossing one leg over other) is normally possible to 150° angle with body. *Internal rotation* is noted by having patient rotate *foot to try to touch toes to opposite heel*—normal rotation 30°. *External rotation* is demonstrated by turning foot outward as if to touch fifth toe to table—normal rotation 60° to 70°. [illegible]
with the patient prone on the table.
is normally possible to about 30° fr [illegible]
extension is nearly always the first movement limited or lost completely. Ankylosis of the hip occurs in various degrees of flexion, usually with

adduction and perhaps internal rotation. *Spasm* or *atrophy* of the gluteal muscles is common with hip disease.

Examination of the Knee.—With the patient standing, observe for

of visible bony landmarks, and lateral bulging of the capsule above and below the patella. *Palpation* reveals the nature of swelling, which may be *localized over the patella in prepatellar bursitis, below the patella in* hypertrophy of the infrapatellar fat pad, or above the patella (under the quadriceps in the supra-patellar pouch) in villous degeneration of the

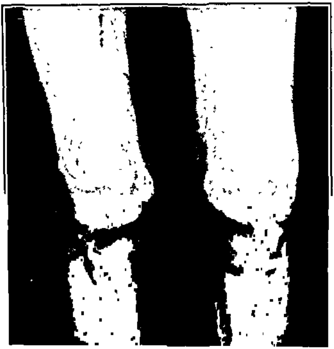


FIG 7.—Chronic rheumatoid arthritis of knees Patella is obscured by thickening of the synovium with effusion, more marked in the left knee

synovium

into the popl

are common

synovial thickening and *effusion* are commonly found in the knee (Fig. 7.)

cooling of an inflamed joint, after artificial heating by diathermy, was slower than in the normal joint, inflamed joints heated up as quickly or more

impaired circulation in the synovial membrane, but this work awaits confirmation.

The most significant finding of these experiments so far is that vascular responses in the joints are *similar* to those of muscles,[12] and apparently *opposite* to that of the overlying skin. Previous hypotheses, based on cutaneous vasomotor reactions alone, supposed that cold, apprehension, and like influences produced a prolonged *ischemia* of joint tissues, predisposing them to arthritis. The above observations, however, would suggest that the opposite might be true—that these same predisposing factors might produce prolonged *synovial congestion*. Further work along these lines may help clarify the part such external influences play in the pathogenesis and perpetuation of arthritis.

An ingenious new yardstick for degree of joint inflammation has been

joint tissues.

Further studies along the lines of the work of Kellgren,[16] Joseph,[14] and others may help to solve some of the many unknown facts about the function of joint tissues. Extensive investigations along the histochemical approach will certainly bring to light much that is still unsolved at this time.

BIBLIOGRAPHY

timore, Wm

272, 1940

8, 409, 1932

Dis 2, 216, 1941

7 BYWATERS, E J L Nature, 138, 288, 1936
8 HENCH, P, BAUER, W *et al* · Ann Int Med 32, 362, 1948
9 HOFFMANN, A, LEAHMANN, G, and WERTHEIMER, E Pfuger's Arch 220, 183, 1928.
10 HOLLANDER, J L and HORVATH, S M: Am Jour Med Sci, 218, 543, 1949
11. HORVATH, S M and HOLLANDER, J L · Jour Clin Invest, 28, 469, 1949
12 HORVATH, S M, MILLER, R, and HUTT, B M.: Am Jour Med Sci · 216, 430, 1948

ability to palpate the bony landmarks—femoral condylar outline, or margin of tibia beside the patella. *Tenderness* should be investigated for severity and location This may be confined to the region of one inflamed *bursa*, may be at the point of *attachment* of the *medial* or *lateral collateral ligaments* to the tibia, about the patellar ligament, or at the tibial tuberosity. (In degenerative changes of the knee secondary to knock-knee and flat feet, the usual point of tenderness is at the attachment of the internal collateral ligament to the tibia. In meniscus injury the point of tenderness is just below the medial femoral condyle). *Motion* of the knee should be checked in *flexion* (normally to 30°), *extension* (normally to 180°), and, with the knee flexed to 90°, *rotation* of the leg on the thigh (normally about 20°). Testing for *abnormal mobility* is important (ligamentous tears—chronic sprains). To check the *external collateral ligament* one of the examiner's hands is placed on the inside of the patient's knee, to make counter-traction to the other hand of the examiner, which pulls the extended lower leg toward the midline. For testing the *internal collateral ligament,*

Crepitation on motion is common in the knee. Coarse crepitation is felt in knees following traum
patellar chondromalacia.
arthritic knee. *Muscle sp*
muscles, *atrophy* is first noticed in the quadriceps. *Contractures* and *ankylosis* usually occur in a position of partial flexion.

Examination of the Ankle.—At this location *periarticular swelling* (edema) from causes other than joint disease is common. Painless, pitting edema of the lower legs and about the ankle is often from old phlebitis, or varicosities if unilateral, or from nutritional, cardiac, or renal deficiency if bilateral. *Swelling* from arthritis of the ankle joint is *tender*, or, in low-grade
he reflections of the synovium of the
Effusion may be demonstrable as
pitation may be noted on motion.
Motion of the *ankle proper* is possible as *dorsiflexion* and *plantar flexion.* Motion of the *sub-astragalar joint* is possible as *inversion* and *eversion*

ful Feet. A few points will be set forth here.

1. With the patient standing, note shape of the foot. *Pronation*, with flattened long arch, is common (*pes planus*). *Splay foot*—spreading and flattening of the metatarsal arch is also common. Note presence of *hallux valgus* (bunion deformity of great toe), *hammer toes* (hyperextension contracture), corns or callouses.
2. *Color* of skin should be noted, and *arterial* (dorsalis pedis and posterior tibial) *pulsations* palpated
3. *Swelling* and *tenderness* should be investigated for severity and location (*e. g.*, over the heel in inflammation of tendo achilles, under heel at attach-

CHAPTER 4

THE SYNOVIAL FLUID

By HOWARD C. COGGESHALL, M.D.

THE articular cavity contains a sticky, pale yellow, clear fluid designated as "synovial fluid" Until recently little attention has been focused on this fluid as a medium for a fuller understanding of joints in health and disease Several investigators[2, 8, 14, 16, 18, 25, 27, 37–40] have made contributions on this subject, and significant studies are being made by Bauer and his associates[4–7, 20–24, 49–55] This latter work is of particular interest because considerable attention has been devoted to the definition of normal fluids in animals as well as humans.[5, 22, 51, 54, 60] Heretofore little information was available because of the difficulty of obtaining sufficient quantities of fluid from normal subjects to make an adequate study Studies based for the most part upon the synovial fluids obtained from dogs and cattle, plus information gained from human subjects, have resulted in enough significant information to establish the characteristics of normal synovial fluid At present, the knowledge of pathological fluids is meager, but it is hoped that its examination will be a more important part of the objective interpr [illegible]

of [illegible]

plasma plus the addition of mucin from the connective tissues and the synovial membrane[5] The chemical constituents and antibodies are essentially the same as blood plasma The protein content is higher because of the presence of mucin and the products of tissue destruction resulting from the normal wear and tear of the normal joint

Anatomy of Articular Structures.—The anatomy of various joints has been studied in great detail[5] In the human embryo the anlage is completely formed early in the third fetal month. There are two chondrogenous plates separated by mesenchymal tissue This tissue becomes liquid and develops into the articular cavity Surrounding this cavity is a connective tissue lining designated as "synovial membrane" This term is anatomical incorrect and should be applied with this understanding. Away from its attachments at the perichondrial and periosteal border, the synovial membrane has a delicate intima which may be separated from a deeper fibrous layer. Unlike articular cartilage, it has unlimited power of regeneration[36, 60] and can be completely replaced within four to eight weeks after surgical removal The synovialis is very vascular and contains a heavy network of lymphatics and nerve tissue. The nutrition of the articular cartilage is derived from the synovial fluid[3, 57] by way of the synovial membrane except for the small amount available from the subchondrium.

The articular cartilage is one of the most primitive tissues and lacks both vascular and nervous tissue. The low oxygen requirement and its limited reparative ability show that cartilage is a relatively inactive tissue. (See

ment of plantar fascia, under heads of metatarsals, over dorsum of foot, or in toe joints). The metatarso-phalangeal (MTP) joints are commonly involved in rheumatoid arthritis, and are also tender in weak feet and other conditions. *Skin lesions* about the feet and toes should be noted—the presence of dermatophytosis, keratosis, psoriatic nail changes, and ulcers

Sample Clinic Work-up Sheet (foreshortened)

ARTHRITIS CLINIC

Name. Date Referred by:

Age Occupation Address:

History Family Incidence
Previous Joint Disease or Injury Operations
Prodomes Precipitating Factors
Present Illness Date of Onset Infections:
Type of Onset
Joints Involved: (In Sequence)
Systemic Reactions
Morning Stiffness Jelling: Exercise Effect·
Night Pain Vesperal Aching Weather Effect.
Previous Treatment and Effects
Course Present Complaints.

Physical Examination Height. Weight Appearance
Eyes Blood Pressure
Nosé and Throat Teeth
Heart· Chest Expansion
Lungs Pelvic or Prostatic
Abdomen Nodules·
Lymphadenopathy. Vascular Changes
Skin changes.

Joints	Tender-ness	Heat	Swelling	Motion in °	Muscle (Spasm) (Atrophy)	Crepita-tion	Other

Pertinent Laboratory Data· Hb RBC WBC: Diff.:
Sedimentation Rates
Blood Uric Acid
Other Studies
Synovial Fluid.
X-ray Changes
Diagnosis
Treatment Recommended

previous chapter) Its poor power of regeneration and repair have been

underlying subchondral bone.[4] The synovial fluid in traumatic conditions

apparent barrier between the articular cavity and the intercellular spaces of the synovialis. Direct communication between the joint space and the lymphatics has not been demonstrated. Substances of small molecular size such as sugar, urea, uric acid, non-protein nitrogen, thiocyanate, mecholyl, pituitrin and pilocarpin are removed by way of the capillaries of the synovia. *Substances of large molecular size and particulate matter such as* proteins, bacteria and blood cells, are removed by way of lymphatics. It has been demonstrated that egg albumen appears in the synovial fluid within five minutes after intravenous injection. Egg albumen and horse serum albumen can be found in the thoracic duct within thirty minutes

Normal Synovial Fluid[1,20,27,55]

Synovial fluid is similar to plasma dialysate and is an example of tissue fluid in general[5]
Amount: 0 1 to 2 0 cc (average 0 45 cc.)
Total leukocyte count: 180 cells or less per c mm (not affected by cytology of peripheral blood).[27]
Average differential pattern.[27]
- Monocytes, 48 per cent.
- Lymphocytes, 25 per cent
- Polymorphonuclears, 7 per cent
- Clasmatocytes, 10 per cent
- Unclassified phagocytes, 5 per cent.
- Synovial cells, 3 per cent.
- *Unclassified cells, 2 per cent.*
- No eosinophils or basophils.

Erythrocytes are not normally present in synovial fluid.
Culture—negative.
Total protein—less than 2 1 per cent,[20] albumin/globulin—4, this varies greatly
... 0 mg per cent, fibrinogen,
(10 mg %).
Mucin—0 55 to 1.10 gm (average 0 85) Mucin is responsible for the osmotic pressure[54]
pH—7 4.
Specific gravity—1 008 to 1 015, average 1 010.
Viscosity—average—150 (varies from 51 to 403)[54]

4

may give diagnostic hints *Excessive sweating* usually accompanies the swelling of the feet from active rheumatoid arthritis.

4. *Movements* of the feet, particularly for joint mobility can be checked [illegible] heads or "stand- [illegible] s, and grasping, [illegible] *Manipulation* of the tarso-metatarsal, and metatarso-phalangeal joints will elicit tenderness if tenosynovitis or arthritis is present. (See also chapter on Painful Feet).

In most patients with joint complaints, the history and a quick survey will reveal which joints are involved, so that attention can be focused at these points. If the examiner quickly observes and palpates the joints while he and the patient carry them through normal range of motion, he can spot which joints are abnormal, and examine them in detail after checking all the others.

USEFUL LABORATORY TESTS IN THE DIFFERENTIAL DIAGNOSIS OF ARTHRITIS

1. *Sedimentation rate* *Normal figures* are obtained in uncomplicated degenerative joint disease (osteo-arthritis, hypertrophic arthritis), in most cases of fibrositis, in the intervals between acute attacks of gout and in subdeltoid bursitis. *Increased sedimentation rates* are usually found in the active stage of rheumatic fever, rheumatoid arthritis, during acute attacks of gout, gonorrheal arthritis and other forms of specific infectious arthritis, rheumatoid arthritis of the spine and in tuberculous arthritis (See Chapter on Sedimentation Rate.)
2. *Blood uric acid* *The only rheumatic disease in which the blood uric acid* [illegible]
3. [illegible] joint symp- [illegible] done free of [illegible] state laboratory. *Physicians should* [illegible]
4. [illegible] be done in all patients with arthritic symptoms Joint pains may be an early sign of *leukemia*, especially in children. *Diabetes mellitus* may mimic the loss of weight, tiredness and pains in the extremities which occur in rheumatoid arthritis. *Anemia* and leukocytosis are common in rheumatoid arthritis Renal impairment is common in gout, and in the diffuse collagen diseases
5. *Basal metabolic rate* is useful in establishing the diagnosis of myxedema or hyperthyroidism.
6. *Roentgen examination* of several of the involved joints is of assistance in diagnosis and prognosis. These investigations may occasionally reveal *evidence of unsuspected pathology—such as metastatic carcinoma.* (See Chapter on Radiology in Arthritis.)
7. *Examination of synovial fluid*, especially its cell count and the differential pattern of the leukocytes, may prove useful. In acute infectious forms of arthritis, the total leukocyte count and the percentage of polys are greatly increased in this fluid; this is also true, to a lesser degree, in rheumatoid arthritis and rheumatic fever. In traumatic arthritis, the cell count is only slightly increased. (See Chapter on Synovial Fluid.)

cavity, if at all. The products of exudation into the joint cavity and tissue necrosis frequently result in an increased protein content and thereby cause an increased osmotic pressure and effusion.

Normal synovial fluid is clear, pale yellow, with a specific gravity of 1 010 to 1 012. The distribution of the electrolytes and non-electrolytes between the serum and the synovial fluid is in accordance with the concept that it is a dialysate and conforms to the Gibbs-Donnan formula for tissue membrane equilibrium. Any substance in the blood plasma which is permeable to the capillary epithelium can be found in the synovial fluid. Chemotherapeutic agents such as sulfonamides, penicillin, sodium salicylate, and various dyes can be readily demonstrated in the synovial fluid in approximately the same quantities as found in the blood plasma. As a rule, the level of these substances does not reach the height of the blood level and there is a lag in the peak of the concentration in the synovial fluid as compared with the blood.[4]

Synovial Mucin —Recent studies indicate that the synovial mucin is denatured by intra-articular infections and rheumatoid arthritis.[54] The normal fluid shows an elastic, ropy precipitate with the addition of acetic acid. Infected fluids (Group III) fail to show this reaction and show only increase in the turbulence. Fluids from patients with rheumatoid arthritis show a similar reaction depending on the severity of disease. Traumatic and rheumatic fever fluids show a normal mucin content whereas in myxedema and osteoarthritis they show an increased amount.

Traumatic effusions have a normal mucin content despite the great increase in fluid volume.[54] A high mucin content is found in osteochondromatosis and in occasional cases of degenerative joint disease, neuropathy, and disseminated lupus erythematosus.[54] Exceptionally high concentrations of mucin can be found in the synovial fluid and subcutaneous tissues of myxedematous patients[47,54] Ord found that the amount of mucin obtained, postmortem, from the skin of a myxedematous patient exceeds a normal control fifty times[47]

[illegible]

tion is of traumatic origin (such as traumatic arthritis, degenerative joint disease and neuroarthropathy) the viscosity is fairly high, the mucin concentration is essentially normal and precipitation with acetic acid produces [illegible] (Table 1.) *Therefore the* [illegible]

mucin is rare in effusions of rheumatic fever and lupus erythematosus disseminatus may aid in their diagnosis. In rheumatoid arthritis and in infectious arthritis of known origin, the fluids tend to be less viscous, the mucin concentration is usually below normal, and the acetic acid precipitate is normally a soft friable mass or merely shreds in a cloudy solution. (Table 1.) *In these diseases, the mucin becomes more normal as the joint involvement subsides, a fact which may be of prognostic value* "[54]

Denatured mucin is the result of hydrolysis caused by various substances (mucinase or hyaluronidase)[49,54] which include bacterial enzymes, the "spreading factor"[17] derived from testicular and tumor extracts, ascorbic acid and hydrogen peroxide.[54]

8 Antistreptolysin and antihyaluronidase titres of serum are helpful in differentiating rheumatic fever and rheumatoid arthritis. Plasma or bone marrow preparations for "L. E." cells are helpful in differentiation of acute disseminated lupus erythematosus

9 *Miscellaneous laboratory examinations* sometimes needed: smear of urethral or cervical discharge for Gc; Gc complement fixation test; agglutination test for Brucellosis; examination of prostatic secretion for leukocytes, x-ray of the teeth, paranasal sinuses and gall bladder for

(elevated in hyperparathyroidism, and hypervitaminosis D).

If special facilities are available for bacteriologic study for pleuropneumonia-like "L" organisms, all cervical, vaginal, prostatic and urethral exudates from cases of arthritis with genito-urinary infection *should have special cultures for this group of infectious agents.*

Synovial mucin is present in the joint and subsynovial tissues[1 51 53] Its concentration varies in normal human synovial fluids from 0 3 to 0.8 gm per 100 cc.[1 11,16 51 53,54] It has been pointed out that this polysaccharide-protein in various combinations are to be found in all tissues of the body They have been isolated from skin,[17 19 40 85] connective tissues,[49] lungs,[28] vitreous humor,[42] aqueous humor,[44] and umbilical cord.[43] Polysaccharide and mucin have been isolated from tissues in the following pathological conditions: Tumors of the pleura,[45] fowl sarcoma,[21 48] myxedematous tissue,[47 51] inflamed pleura in a patient with rheumatoid arthritis,[54] and the encapsulated phase of strains of group A and C hemolytic streptococci.[30 41 56] The wide-spread occurrence of mucin in the various tissues indicates that its elaboration is not confined to the synovial membrane and subsynovial tissues.

synovial fluid leukocytes indicates that they are similar to that of tissue

mononuclear cells and, for that reason, it is best to designate the cells as "mononuclear cells" and "polymorphonuclear cells" The stain should be

cytes *The polymorphonuclear cells should not exceed 25 per cent of the synovial fluid leukocytes in normal fluid.* Transudates and traumatic effusions have low total leukocyte counts and approximately normal differential counts. The reverse is found in exudative fluids associated with infections and conditions which simulate infections, *i e*, rheumatic fever, rheumatoid arthritis and gouty arthritis. These exudates are characterized by an increase in the total leukocyte count and the percentage of polymorphonuclear leukocytes Tuberculous synovitis is the only exception to this statement Tuberculous infections may show a leukocytosis without an elevated percentage of polymorphonuclears

Pathological Synovial Fluids.—Bauer and associates[20 23 50–55] have divided pathological synovial fluids into 3 groups Group I consists of the *Traumatic Fluids* which resemble normal synovial fluid. Group II, *Intermediate*, and Group III, *Septic Fluids*. Trauma causes an increase in the amount of fluid with little change in the ch…
content.
polymorph
Group I include traumatic effusions, Charcot joints and degenerative joint disease (osteoarthritis)

Group III, *Septic*, is composed of fluids which have similar characteristics to those obtained from septic or infected synovial fluid There is evidence

CHAPTER 3

THE STRUCTURE AND FUNCTION OF ARTICULAR TISSUES

By Joseph L. Hollander, M.D.

This discussion is an attempt to bring together some of the essential

together are joints. Thus the suture lines of the skull are joints, where the adjoining bone margins are fixed by a bridge of fibrous tissue. The symphysis pubis is a "fixed" joint or *synchondrosis*, in which the bone ends are bridged by tough fibrocartilage and ligaments allowing little movement. The articulations between the bodies of the vertebrae represent another type of joint in which the bony surfaces are separated by a cushion of fibrocartilage (the intervertebral disks), and are joined by dense fibrous bands or ligaments *Diarthroses*, the joints which permit free movement, are the ones most prone to injury and disease. They differ from the above

tissue is surrounded and supported by a *capsule* of white fibrous connective tissue, reinforced by *ligaments*. In certain of the joints, such as the knees, temporomandibular and sternoclavicular joints, there is a partial or complete interposing layer of fibrocartilage between the hyaline cartilages.

These elementary anatomic facts about joints have been known for centuries, but, until relatively recently, little has been learned of the physiology of the articular tissues. Even now the knowledge of the processes of growth, aging, nutrition, innervation, circulation, and even of the mechanical functions of joints is scanty and incomplete, but an increased interest in the basic physiology of the joints on the part of investigators is slowly bringing to light a few new facts concerning the above mechanisms.

Much of the investigation of joint physiology has been on the origin, characteristics, and disposition of the constituents of the synovial fluid. This knowledge is described in the next chapter (The Synovial Fluid). What is known of the aging process in joint structures is described in Chapter 38. In the present discussion, major attention is given to the articular cartilage and its characteristics, and to certain vascular and neuromuscular mechanisms as they pertain to joint function.

Embryologically and *histologically*, the tissues making up a typical diarthrosis are of mesenchymal character. The *synovial membrane* is

upon the addition of acetic acid. Ropes, *et al.*,[54] have demonstrated that this abnormality is caused by hydrolysis of mucin. Septic fluids should be proved by positive cultures The most common conditions found in Group III are tuberculous, gonorrheal, septic (streptococcic, staphylococcic, etc.,) arthritis

Group II (*Intermediate*), have characteristics of both the traumatic and septic fluids The effusions of rheumatic fever, gouty arthritis and intermittent hydrarthrosis are at first acute with an inflammatory exudative reaction and then subside with a gradual return to normal. If the fluid is examined early, it will resemble a septic fluid with a high leukocyte count and a preponderance of polymorphonuclears. Later, as the effusion subsides, there is a decrease in the leukocyte count and polymorphonuclears with a predominance of mononuclear cells and lymphocytes. Therefore the findings in Group II will depend entirely upon the duration of the effusion and the severity of disease. In acute rheumatic fever, the return of the cytology to normal is prompt after the administration of adequate amounts of salicylates[24] The synovial fluid of patients with rheumatoid arthritis may have characteristics of the intermediate and septic groups. Even when the disease is mild and the leukocyte count is low, there is usually an increase in the polymorphonuclear cells. In severe rheumatoid

mucin with the acetic acid test, increased clot formation, decreased viscosity, and a marked reduction of the fasting sugar content.

Cytologic Technic for Synovial Fluid.—The examination should always be made on a synovial fluid specimen containing an anticoagulant.[22] The most convenient procedure is to use an oxylated bottle that is used for collecting blood sugar. Care should be taken to agitate the oxylated fluid to prevent coagulation because the sodium oxylate does not enter the

count should be done under high power using the portion of the chamber marked for the red blood cell count. A rough differential count can be made on the same specimen if methyl violet has been added to the saline

to over-stain the preparation.

The fixed smear method will differentiate the polymorphonuclear leukocytes from the mononuclear cells, however, it will not accurately differentiate the types of mononuclear cells. In order to distinguish the monocytes, mononuclear phagocytes and lymphocytes, supervital stains with neutral red and Janus green must be employed.[22] The most important diagnostic information comes from the percentage of polymorphonuclears, therefore it is not necessary to differentiate the various types of mononuclear cells. For this reason, the fixed smear method is more practical for the usual clinical examination.

intimal layer of the synovial membrane the cells are abundant, forming layers or aggregations, but in the tissues more remote from the joint space

cavity, but more accurately a *tissue space* This concept makes it easier

connective tissue cells and fibers.

cartilaginous surfaces, providing lubrication. A crude analogy can be

interposed between the steel surfaces. The articular cartilage, likewise, is a buffer—softer and more adaptable than bone to changes in contour, yet resilient and elastic enough to resume its original contour when the strain of compression is removed

The importance of the film of synovial fluid maintained between the articulating cartilages was demonstrated by Jones,[13] when he found that the coefficient of friction in a dry joint was 14 times as great as in a joint kept lubricated by the synovial fluid In his experiment, the stifle joint of a horse was removed from the carcass and set up in an apparatus which mechanically compressed and flexed the joint. If synovial fluid was present, no damage to the joint surface was observed even after several hours of mechanical motion, but when the fluid was removed, marked heating was noted, and the articular surfaces quickly became rough and worn down. This "friction effect" in joint tissues after exercise was borne out by *observations of Hollander and Horvath,[10,11] in studies on joint temperature* variations. Even in normal joints the internal articular temperature

TABLE 1

NORMAL AND PATHOLOGICAL SYNOVIAL FLUID

Minimum and Maximum Values With the Average in Parenthesis

A composite Table made from published and unpublished data by Bauer and Associates[4-8 21-24 50-55] †

	Synovial Fluid Group	Joint Disease	Appearance	Clot Form.	Leukocytes Cmm	Polys. %	Viscosity	Protein (Gm)	Globulin (Gm)	Mucin (Gm)	Sugar* Mg %
Normal		Normal[12]	Clear	0	13-180 (63)	0-25 (7)	51-403 (203)	0 23-2 13 (1 36)	0 05	0 55-1 10 (0 85)	0-20 (7)
Normal	I Traumatic	Traumatic Arthritis	Clear to bloody	0-2 plus	[illegible]	0-36 (10)	15-34 (37)	3 2 -5 1 (4 0)	0 7 -1 4 (0 8)	0 35-1 57 (0 73)	0-20 (7)
Normal	I Traumatic	Charcot Joint	Clear or turbid	0-1 plus	[illegible]	0-45 (17)	32-228 (09)	1 8 -5 1 (3 3)	0 3 -1 7 (1 1)	0 07-1 61 (0 59)	0-19 (10)
Normal	I Traumatic	Degenerative Jt Dis (Osteoarthritis)	Clear	0-2 plus	[illegible]	0-58 (8)	4-363 (88)	1 8 -4 9 (3 2)	0 3 -1 4 (0 8)	0 23-1 03 (0 67)	6
Exudate	II Intermediate	Gouty Arthritis	Turbid	1-2 plus	[illegible]00	48-94 (83)	4-6 (5)	3 1 -5 0 (4 3)	1 1 -1 8 (1 4)	0 28-0 80 (0 53)	0-41 (12)
Exudate	II Intermediate	Rheumatic Fever	Slightly turbid	0-3 plus	[illegible]00	8-98 (46)	31-50 (38)	1 6 -4 9 (3 5)	0 4 -1 1 (0 7)	0 64-1 21 (0 87)	6
Exudate	II Intermediate	Rheumatoid Arthritis	Clear to turbid	0-3 plus	[illegible]00 [illegible])	5-96 (65)	4-116 (17)	3 0 8 9 (4 9)	1 2 -6 8 (2 2)	tr -1 35 (0 52)	0-88 (31)
Exudate	III Septic	Tuberculous Arthritis	Turbid	0-2 plus	[illegible]00 [illegible])	29-96 (67)	13	4 1 -6 1 (5 1)	1 4	0 41-1 19 (0 62)	0-108 (57)
Exudate	III Septic	Gonorrheal Arthritis	Turbid	0-3 plus	[illegible]00 [illegible])	2-96 (65)	3-21 (11)	4 3 -6 9 (5 4)	1 31-3 1 (2 1)	0 13-1 20 (0 49)	0-97 (26)
Exudate	III Septic	Septic Arthritis	Purulent	2 plus-3 plus	[illegible]00	75-100 (95)	25-38 (33)	3 7 -6 9 (5 0)	1 4 -2 2 (1 9)	tr.-0 58 (0 40)	40-122 (71)

* Difference between the Blood and Synovial Fluid

† Presented before the American Rheumatism Association in Chicago (1948). Ropes, M.

tion beyond which the matrix is calcified and fixed to the subchondral dense bone. The cartilage contains no blood vessels, lymphatics, or nerves.[2]

Resilience is one of the prime characteristics of articular cartilage. It is almost completely elastic to frequent intermittent pressure, while continuous compression tends to slow the rate of recovery to its original shape. The

cartilage which have been broken off and float free in the joint may remain viable for long periods [3]

The respiration of cartilage is nearly nil.[7] Like muscle, it possesses the faculty of anaerobic oxidation.[2] The metabolic rate is only one-tenth that of connective tissue, apparently because of the great preponderance of matrix over cells Studies have shown[9] that young cartilage contains many

complexes of hyaluronic acid, lactates, and calcium salts.[8]

Cartilage cells grow by mitosis when the cartilage is immature. In adult cartilage amitosis is the sole form of growth. Proliferation of cartilage following injury is greater in the deeper zones [8] Apparently there is some influence on cartilage growth and repair by anterior pituitary hormones. In young animals such hormones induce hypertrophy in the transitional zone, accompanied by liquifaction and ulceration of the cartilagenous surface, but in adult animals only these degenerative changes were seen. Thyroid extract increased cartilage growth in young animals.[17] Apparently articular cartilage requires apposition with the opposite cartilage of the joint to maintain its integrity, since it regenerates better in weight bearing areas than in non-weight bearing regions [3] The aging processes of cartilage are discussed fully in Chapter 38

decline in oxygen activating systems because oxygen consumption in the presence of methylene blue seemed independent of age Bauer[8] believes that this methylene blue reaction for determining oxygen consumption has nothing to do with actual tissue respiration, but the fact is still established that older cartilage has a much lower respiratory exchange than young tissue.

and peri-articular tissues, and apparently are the receptors for *pain.*

of exudation with a marked increase in the leukocytes, granulocytes, and the total protein. The sugar content is reduced due to the metabolic activity of the leukocytes and/or bacteria. There is a failure of mucin to precipitate upon the addition of acetic acid Ropes, *et al.*,[11] have demonstrated that this abnormality is caused by hydrolysis of mucin. Septic fluids should be proved by positive cultures. The most common conditions found in Group III are tuberculous, gonorrheal, septic (streptococcic, staphylococcic, etc.,) arthritis.

Group II (*Intermediate*), have characteristics of both the traumatic and septic fluids. The effusions of rheumatic fever, gouty arthritis and intermittent hydrarthrosis are at first acute with an inflammatory exudative reaction and then subside with a gradual return to normal. If the fluid is examined early, it will resemble a septic fluid with a high leukocyte count and a preponderance of polymorphonuclears. Later, as the effusion subsides, there is a decrease in the leukocyte count and polymorphonuclears with a predominance of mononuclear cells and lymphocytes. Therefore the findings in Group II will depend entirely upon the duration of the effusion and the severity of disease. In acute rheumatic fever, the return of the cytology to normal is prompt after the administration of adequate amounts of salicylates [24] The synovial fluid of patients with rheumatoid arthritis may have characteristics of the intermediate and septic groups. Even when the disease is mild and the leukocyte count is low, there is usually an increase in the polymorphonuclear cells. In severe rheumatoid

mucin with the acetic acid test, increased clot formation, decreased viscosity, and a marked reduction of the fasting sugar content.

Cytologic Technic for Synovial Fluid.—The examination should always be made on a synovial fluid specimen containing an anticoagulant.[22] The most convenient procedure is to use an oxylated bottle that is used for collecting blood sugar. Care should be taken to agitate the oxylated fluid to prevent coagulation because the sodium oxylate does not enter the solution readily. The fluid is then counted with the white cell pipette using physiological saline as the dilutant. Acetic acid solution used for the white blood count should not be used because it precipitates the mucin. The count should be done under high power using the portion of the chamber marked for the red blood cell count A rough differential count can be made on the same specimen if methyl violet has been added to the saline dilutant. Differential counts should also be made by staining fixed smears prepared on coverglasses with Wright's or Giemsa's stain. The coverglass smears are preferred because the film is thinner. Care should be taken not to over-stain the preparation.

The fixed smear method will differentiate the polymorphonuclear leukocytes from the mononuclear cells, however, it will not accurately differentiate the types of mononuclear cells. In order to distinguish the monocytes, mononuclear phagocytes and lymphocytes, supervital stains with neutral red and Janus green must be employed [22] The most important diagnostic information comes from the percentage of polymorphonuclears, therefore it is not necessary to differentiate the various types of mononuclear cells. For this reason, the fixed smear method is more practical for the usual clinical examination.

Paccinian corpuscles are ovoid bodies *consisting of concentric fibrous tissue* layers over a central nerve filament which are believed to be the special

muscles, bones, and perhaps the skin of the same area. The sensory

same nerve trunks that supply the joint also supply the muscles moving that joint, and the skin areas over the insertion of those muscles [19] The sensory innervation of a joint seems so constituted that, as part of the capsule is tightened or stretched as the joint approaches its limit of movement, a reflex contraction of the antagonist muscle is produced This protective reflex prevents undue wear and tear on the capsular structures from sudden or unguarded movements Joint movement is also controlled by a reciprocal innervation between the groups of muscles responsible for motion of the articulation. As a joint flexes, the extensor muscles do not

as neurosyphilis, leprosy, and syringomyelia, that the Charcot joint develops—a marked degenerative process, though not necessarily a painful one. It is in part from over-irritability of these reflexes that muscle spasm is produced by inflammation of the articular tissues.

Joint pain results from any agent irritating the free nerve endings—whether from inflammation and increased pressure in the tissue, or from *trauma Recent studies by Horvath and Hollander*[11] *have confirmed* the the extreme sensitivity to pain of the capsular and synovial tissues, yet absence of pain sensation in the cartilage and perhaps even the subchondral bone. In their studies of intra-articular temperature, joint aspirations were performed on many normal and inflamed joints. If any portion of the joint capsule or synovial membrane was traumatized by the aspirating needle, or the thermocouples inserted through them, a sharp pain was

localized.

Kellgren[18] has recently performed experiments on the peripheral genesis of pain. He produced pain by chemical changes, thermal changes, or circulatory changes in the part. Conclusions were not definite, since it was found that any of the agents could produce pain, but the type of pain varied greatly with the agent used. In most cases of chronic arthritis,

TABLE 1

NORMAL AND PATHOLOGICAL SYNOVIAL FLUID

Minimum and Maximum Values With the Average in Parenthesis

A composite Table made from published and unpublished data by Bauer and Associates[4-8,21-24,50-55]†

	Synovial Fluid Group	Joint Disease	Appearance	Clot Form	Leukocytes Cmm	Polys %	Viscosity	Protein (Gm)	Globulin (Gm)	Mucin (Gm)	Sugar* Mg %
Normal	I Traumatic	Normal[12]	Clear	0	13-180 (63)	0-25 (7)	51-403 (203)	0 23-2 13 (1 36)	0 05	0 55-1 10 (0 85)	0-20 (7)
Normal	I Traumatic	Traumatic Arthritis	Clear to bloody	0-2 plus	[illegible]	0-36 (10)	15-34 (37)	3 2 -5 1 (4 0)	0 7 -1 4 (0 8)	0 35-1 57 (0 73)	0-20 (7)
Normal	I Traumatic	Charcot Joint	Clear or turbid	0-1 plus	[illegible]	0-45 (17)	32-228 (99)	1 8 -5 1 (3 3)	0 3 -1 7 (1 1)	0 07-1 61 (0 59)	0-19 (10)
Normal	I Traumatic	Degenerative Jt Dis (Osteoarthritis)	Clear	0-2 plus	[illegible]	0-58 (8)	4-363 (88)	1 8 -4 9 (3 2)	0 3 -1 4 (0 8)	0 23-1 03 (0 67)	6
Normal	II Intermediate	Gouty Arthritis	Turbid	1-2 plus	[illegible]	48-94 (83)	4-6 (5)	3 1 -5 0 (4 3)	1 1 -1 8 (1 4)	0 28-0 80 (0 53)	0-41 (12)
Exudate	II Intermediate	Rheumatic Fever	Slightly turbid	0-3 plus	[illegible]	8-98 (46)	31-50 (38)	1 6 -4 9 (3 5)	0 4 -1 1 (0 7)	0 64-1 21 (0 87)	6
Exudate	II Intermediate	Rheumatoid Arthritis	Clear to turbid	0-3 plus	[illegible]	5-96 (65)	4-116 (17)	3 0 8 9 (4 9)	1 2 -6 8 (2 2)	tr -1 35 (0 52)	0-88 (31)
Exudate	III Septic	Tuberculous Arthritis	Turbid	0-2 plus	[illegible]	29-96 (67)	13	4 1 -6 1 (5 1)	1 4	0 41-1 19 (0 62)	0-108 (57)
Exudate	III Septic	Gonorrheal Arthritis	Turbid	0-3 plus	[illegible]	2-96 (65)	3-21 (11)	4 3 -6 9 (5 4)	1 31-3 1 (2 1)	0 13-1 29 (0 49)	0-97 (26)
Exudate	III Septic	Septic Arthritis	Purulent	2 plus-3 plus	[illegible]	75-100 (95)	25-38 (33)	3 7 -6 9 (5 0)	1 4 -2 2 (1 9)	tr -0 58 (0 40)	40-122 (71)

* Difference between the Blood and Synovial Fluid

† Presented before the American Rheumatism Association in Chicago (1948). Ropes, M

arterial occlusion and venous congestion had no direct effect in producing pain, but cooling had a definite effect. In acute joint inflammation, however, circulatory change had a pronounced effect on pain in the joint. Lowered pain threshold was found in such conditions, also, and pain could be produced by chemical change, increased tension in the tissues, ischemia, or by temperature changes.

Why changes in the weather so commonly increase joint pain has never been adequately explained.[15] It may be *hypothesized* that impaired function in maintaining equilibrium between the intercellular and intracellular fluids during changes in external atmospheric conditions may produce temporarily increased *relative* pressure at the pain end-organs. Studies of intercellular osmotic pressures under changing environmental conditions may shed light on the increased joint pain accompanying weather changes experienced by many rheumatic patients.

Circulation in Articular Structures.—As stated elsewhere, the synovial membrane is richly supplied with blood vessels and lymphatic channels. The synovial fluid, a dialysate of blood plasma, apparently carries both nourishment and oxygen to the articular cartilage, which has no vascular supply (see also chapter on Synovial Fluid). Until recently, little direct attention has been paid to the changes in circulation of the synovial membrane produced by disease or external influences, or on the vaso-motor reactions of the synovial vessels By simultaneous determinations of intra-articular temperatures and skin temperatures over the joint, Horvath and Hollander[10,11] *have studied some of the synovial reactions produced by* [illegible] apprehension, pain, the joint (implying [illegible] nfluences decreased skin temperature (cutaneous vaso-constriction). Heat, and certain other influences which produced increased skin temperature *depressed* the joint temperature, at least temporarily When the external stimulation was removed, there was a positive reflex effect manifested by a temperature [illegible]

synovial vaso-motor reactions has been borne out in repeated observations under various conditions. The magnitude of the reflex response in the [illegible]

the cooling curve they estimated that the normal circulation rate in articular structures in dogs was 0 75 cc per minute per cc of static fluid. The circulation rate determined by this method was found to vary with external changes of temperature and administration of drugs This work [illegible]

mation, and was more closely coordinated with activity of the joint inflammation than skin temperature over inflamed joints. While the rate of

Method of Aspirating Joint Fluid.—Surface skin over the joint is cleansed with soap and water, iodine and alcohol in order. Sterile towels are draped in such a manner that the field is aseptic. The procedure should be considered as a surgical one and strict asepsis should be observed throughout, contamination of the joint cavity must be guarded against. The skin overlying the area of puncture, subcutaneous tissues, deeper tissues and joint capsule are infiltrated with 2 per cent procaine hydrochloride. It is preferable to use a short beveled lumbar puncture type of needle in the aspiration of fluid from joints, the stylet should not be removed until the joint space has been entered A large bore needle (16 or 18 gauge) is advised; this will not become clogged by thick exudative material. The joint fluid may be removed by suction with a 50 cc. syringe

[illegible]

desirable to inject air for visualization of the joint space or for therapeutic purposes, it may be done by withdrawing air through the side tip into the syringe and then injecting this into the joint cavity.[50] Other forms of apparatus for inflation of joints have been described.[8,9] Various authors have discussed the technic and value of arthroscopy,[10–12] joint biopsy and visualization of joints by injection of drugs.[33–35]

The technic for aspiration of specific joints is described in Chapter 18 (Intra-articular Injection of Hydrocortisone).

A Practical Routine Examination of Joint Fluid Should Include:

Appearance; quantity; turbidity; reaction, specific gravity, clot formation; viscosity and icterus

Total leukocyte count

Total erythrocyte count.

Differential count (per cent of polymorphonuclear and mononuclear cells).

Chemistry. Total proteins, sugar (fasting).

Viscosity and character of mucin with acetic acid precipitation.

Culture, aerobic and anaerobic if infection is suspected

Guinea pig inoculation if tuberculosis is suspected.

Icterus index if traumatic arthritis is suspected.

Gram stain of centrifuged sediment.

BIBLIOGRAPHY

1 Achard, C and Piettre, M Compt rend Acad d sc 191, 1412, 1930
2 Adkins, E W O, and Davies, D V Quart Jour Exper. Physiol, 30, 174, 1940
3 Allison, N. and Ghormley, R K. Diagnosis in Joint Disease, Baltimore, William Wood & Co, 1931
4 Bauer, W Jour Am Med Assn, 117, 1648, 1941.
5 Bauer, W, Ropes, M W and Waine, Hans Physiol Rev 20, 272, 1940
6 Bauer W, and Short, C L New England Jour. Med, 223, 286, 1940
7. Bennett, G H, Bauer, W, and Maddock, S J Am J Path, 8, 490, 1932
8. Berg, R F Am Jour Surg, 8, 1277, 1930
9 Bernstein, M A, and Arens, R A Radiology, 7, 500, 1926.
10. Burman, M. S Jour Bone and Joint Surg, 13, 669, 1931
11. ———: Arch Surg, 32, 524, 1936
12. ——— Acta Rheumatol, 9, 10, 1937.
13 Burman, M S, and Mayer, L Arch Surg, 32, 846, 1936

14. Bywaters, E G L: Reports on Chr Rheum Dis No 2, London, H K Lewis & Co, Ltd, 1936.
15 Cajori, F A, Crouter, C Y., and Pemberton, R. Arch. Int Med, 37, 92, 1926
16 Cajori, F A, and Pemberton, R. J Biol Chem, 76, 471, 1928
17 Chain, E · Brit J. Exper. Path, 21, 324, 1940
18 Cherry, J H, and Ghormley, R K.: Jour Bone & Joint Surg, 20, 48, 1938
19 Claude, A J Proc Soc Exp Biol & Med, 43, 684, 1940
20 Coggeshall, H C, Ropes, M W, Rossmeisel, E C, Bauer, W.: Jr Clin Invest, 16, 675, 1937
21 Coggeshall, H C, and Bauer, W. New England Jour Med, 220, No. 3, 85, 1939
22 Coggeshall, H. C, Warren, C. F, and Bauer, W.· Anat Rec, 77, 129, 1940
23 Coggeshall, H C, Bennett, G A, Bauer, W: Am Jour. Med Sci, 202, 486, 1941
24 Coggeshall, H C · Texas State Jour Med, 42, 430, 1946
25 Collins, D H · Jour Path & Bact, 42, 113, 1936
26 Fisher, A G T Lancet, 2, 541, 1923
27 [illegible] 35, 740, 1936
28 [illegible]
29 [illegible]
30 [illegible] J Biol Chem, 118, 61, 1937
31 Keefer, C S, Myers, W K, and Holmes, W F., Jr Arch Int Med, 54, 872, 1934
32 Keefer, C S, and Spink, W. W · Jour Am Med Assn, 109, 1448, 1937.
33. Keller, H New York Med Jour, 100, 458, 1914.
34 ——— Proc Soc. Exp Biol & Med, 27, 850, 1930
35 ——— Med Rec, 141, 76, 1935
36 Key, J A Jour Bone & Joint Surg, 23, 793, 1925
37 [illegible]
[illegible]
40 [illegible]
[illegible]
44 ——— Am J Ophthalmol, 19, 859, 1936
45. ——— J Biol Chem, 133, 83, 1940
46 ———. J Biol Chem, 138, 491, 1941
47 Ord, W M Med Chir Trans, 61, 57, 1878
48 Pirie, A Brit J Exper Path, 23, 277, 1942
49 Robertson, W V B, Ropes, M W, and Bauer, W J Biol Chem, 133, 261, 1940
50 Ropes, M Am Jour Physiol, 113, 112, 1935
51 Ropes, M, Rossmeisel, E C, and Bauer, W · J Clin Investigation, 19, 795, 1940.
52 Ropes, M, and Bauer, W. Jour Am Med Assn, 113, 1160, 1939
53 Ropes, M, Bennett, G A, and Bauer, W Jour Clin Invest, 18, 351, 1939
54 Ropes, M, Robertson, W V B, Rossmeisel, E C, Peabody, R B, and Bauer W Acta Med Scand, Suppl, 196, 1947
55 Ropes, M, Coggeshall, H C, and Bauer, W.· J Clin Invest, 20, 455, 1941
56 Salkowski, E Virchow's Arch 131, 304, 1893
57 [illegible] /, 1909
[illegible] Jour Path, 11, 953, 1935

30. MILLES, H L, and SALT, H B: Ann Rheumat Dis, 2, 192, 1941.
31. MUSGROVE, 1703 Cited by Jones, R L · Bristol, John Wright & Sons, Ltd, 1909
32 MYER, KARL· Ann Rheum Dis, 7, 37, 1948
33 NICHOLS, R E: Jour Lab & Clin Med, 27, 1317, 1942
34 OPPEL, T W., MYERS, W. K, and KIEFER, C. S · J Clin Invest, 12, 29, 1933
35 RACE, J. Proc Roy. Soc Med, 22, 15, 1929.
36. RAWLS, W. B, GRUSKIN, B J, RESSA, A A and JORDON, M· J. Lab & Clin Med, 19, 830, 1934
37. ROURKE, M D, and ERNSTENE, A C: J Clin Invest, 8, 545, 1930
38 ROURKE, M D, and PLASS, E D J Clin. Invest, 7, 365, 1929.
39 SHEDLOVSKY, THEO, and SCUDDER, JOHN Jour Exp Med, 75, 119, 1942
40 SHAPIRO, M. J · Journal Lancet, 61, 481, 1941.
41 SHORT, C L, DIENES, L, and BAUER, W Jour Am. Med. Assn, 108, 2087 1937.
42 SLOCUMB, C H.: Jour Lab and Clin Med, 18, 711, 1933
43 STAINSBY, W. J, and NICHOLLS, EDITH· Jour Clin Invest, 12, 1041, 1932
44 STEINBERG, C L · Am Jour. Med. Sci, 190, 98, 1935
45 WASSON, V P, and BROWN, E E · Jour Pediat, 23, 24, 1943
46 WEISS, A: Am Jour Med. Sci, 181, 379, 1931.
47 WESTERGREN, A Acta med Scand, 54, 247, 1920
48 WHITBY, L E H, and HYNES, M Lancet, 2, 249, 1938
49 WHITTINGTON, R B, and MILLER, A K Brit Jour Exper Path, 23, 56, 1942 Proc Roy Soc B, 131, 183, 1942
50 WINTROBE, M M · Clinical Hematology, Lea & Febiger, Phila, Ed. 3, 1951

CHAPTER 5

THE SEDIMENTATION RATE

By HOWARD C COGGESHALL, M.D.

THE sedimentation rate is a very important laboratory examination available for the recognition and prognosis of the various arthritides. Frequently, it is the only laboratory abnormality to be found in individuals suffering from rheumatic fever or rheumatoid arthritis. Even though the test is a non-specific one, it frequently gives a definite diagnostic lead in distinguishing the various forms of arthritis, especially in the early stage. This test is most useful in the diagnosis and evaluation of severity of disease in rheumatic fever and rheumatoid arthritis, but it need not be ele-

[illegible]

ie usefulness of [illegible] that this phenomenon has been recognized for many centuries.[29,41] The Greeks recognized that blood collected from sick patients separated into four distinct layers It was thought that these humors which consisted of the dark clots at the bottom of [illegible] ie oxygenated red cell layer (sanguine), [illegible] the supernatant serum (the choleric), [illegible] Musgrove,[31] 1703, observed the buffy clot in the blood of rheumatic subjects. John Hunter[21] recognized that the increased sedimentation was associated with the granularity of the blood which was later described by Lister[25] and Hodgkin[26] as rouleaux formation. Fahraeus,[9 10 11] in 1918, was the first to make a study of the sedimentation rate using an anticoagulant and a simple technic to establish the normal limits of the procedure. This application

[illegible]

normal suspension of erythrocytes is dependent upon the hydrophilic property of their surface layers and their negative electrical charges.[33 39,49]

The phenomenon causing rouleaux formation is poorly understood and the sedimentation rate is really an ind [illegible] e size of these rouleaux masses.[29] This formation is easily distu [illegible] aking or by dilution of the plasma with electrolytes such as s [illegible] ride or citrate. The tendency of the cells to f [illegible] uleaux see [illegible] ttraction rather than

CHAPTER 6

RADIOLOGY IN RHEUMATIC DISEASES

By CHARLES J. SMYTH, M.D.

THE different forms of arthritis may manifest the same basic alterations as detected roentgenographically. The pathologic response of the joint tissues to various stimuli results in the following changes in the roentgenograms: Periarticular swelling, osteosclerosis, osteoporosis, decrease in intra-articular (interosseous) space, changes in bone contour (proliferation or destruction), enlargement of epiphyses, ankylosis, subluxations and deformities. These roentgen findings or various combinations of them occur sooner or later in most types of arthritis and if interpreted with the clinical and laboratory data, are valuable in diagnosis and management. Roentgen findings may, however, be negative in the early stages of joint disease. Even though articular pain, tenderness and slight swelling may be present, the roentgenologist may find none of the above criteria of arthritis; in other words, it is too early to find alterations in the roentgen appearance. Such negative roentgenograms are often seen in early acute gout, serum sickness, or acute rheumatic fever (even though the joint is red, tender and presents a slight swelling).[22] In one series of roentgenograms of more than 500 joints in 60 patients with chronic polyarthritis, 25 per cent showed no roentgen findings although pain or deformity was or had been present.[105]

It is the duty of the clinician to furnish his roentgen colleague with a brief history of the case, noting the age of the patient, the sex, the occupation, the joints affected, the appearance of the joints clinically, the course of the disease, and other salient features. A trustworthy roentgenologic diagnosis must depend on a correlation of these clinical aspects with the roentgenographic findings If the roentgen diagnosis does not agree with the clinical impression, the clinician must not feel that the roentgen diagnosis is final by any means and should go over the films with the [illegible]

[illegible] ...ary path[illegible] lesions.[illegible] ...yroidism, neoplasms in various viscera, or metastases to bone, psoas abscess, Paget's disease, pituitary tumors, spinal abnormalities, scoliosis, multiple myeloma, scurvy, rickets, etc.

Roentgen Examination of Joints.—Practically all of the tissues of joints [illegible]

a fusion because they can be easily disengaged. The cause for the rouleaux formation is the result of changes in the *plasma* rather than in the cells, themselves If specimens of blood showing excessive rouleaux formation are washed free of the plasma, they lose this activity if they are again suspended in normal plasma or saline.

The addition of human plasma fibrinogen or serum globulin to saline suspensions of red cells causes rouleaux formation. An increase in the concentration of these proteins accelerates the settling velocity of the rouleaux masses[17] The simplicity of the test for determining the sedimentation rate of red cells makes available a semi-quantitative method [illegible]

rate even though the fibrinogen concentration is normal.

Certain of the abnormal proteins such as gelatin, nucleic acid and [illegible]

linear colloids. It has been suggested that in rheumatic diseases these linear colloids of the connective tissue might enter the blood stream.[22] [illegible]

[illegible]

shrinkage effect produced by potassium oxylate can be counteracted by the addition of ammonium oxylate which tends to increase the red cell size [illegible]

ever marked changes in the room temperature will effect a change.

The length of the sedimentation tube depends upon the method used and, in all methods, the level of the red cell column is recorded according to time and millimeters of fall There are three definite periods noted: The [illegible]

begin to pack in the bottom of the tube and is referred to as "period of packing."

Another factor which should be taken into consideration is that the sedimentation tube should be *vertical* in order to prevent the red cells from sliding down along the inclined plane of the wall of the tube The tube should be at least 2 5 mm. in diameter and the blood specimen should be carefully shaken to break up the rouleaux formation prior to setting up the test.

ning of the cartilage is seen on the x-ray by a diminution of the joint space. The true joint space has been demonstrated roentgenographically on several occasions.[44, 137] No visualization of the capsule or synovial membrane results unless very soft rays are used and then only in the larger joints

Other roentgen criteria of joint disease include regional bones and particularly the articular co [illegible]
most dense, this is [illegible]
cancellous ends of th [illegible]
cancellous ends.[113]

[illegible] criteria of arthritis is change [illegible]

detecting early osteoporosis and also to confirm any x-ray appearance of subsequent progress.

Special Methods.—Boyd[15] observed the interior of the knee joint visualized with 30 per cent iopax, dissolved in 0 5 per cent procaine. The iopax was quickly absorbed Other dyes have been used to produce satisfactory [illegible] of joints [illegible] and skiodan).[89] Such [illegible]

stereoscopic AP and plain lateral roentgenograms, using soft tissue detail technique Release the air, withdraw the needle, and apply a sterile dressing and elastic bandage

This procedure is not to be undertaken without considerable experience upon cadavers and should always be performed with extreme caution.

To discover traumatic lesions of knees special techniques are recommended to demonstrate the intercondylar notch and certain aspects of the patella.[22] Special views of roentgenography of the spine have been described to visualize the apophyseal joints and the intervertebral foramina.[89]

Radiologic Diagnosis in Arthritis.—No classification of rheumatism based on radiological grounds alone is justified The x-ray changes in joint

CHAPTER 5

THE SEDIMENTATION RATE

By Howard C. Coggeshall, M.D.

The sedimentation rate is a very important laboratory examination available for the recognition and prognosis of the various arthritides. Frequently, it is the only laboratory abnormality to be found in individuals suffering from rheumatic f[illegible]
test is a non-specific one, i[illegible]
distinguishing the various f[illegible]
This test is most useful in [illegible]
ease in rheumatic fever anc[illegible]
vated to make a diagnosis in these two conditions.

Before proceeding with further discussion concerning the usefulness of the sedimentation rate, it seems worth while to point out that this phenomenon has been recognized for many centuries.[29,41] The Greeks recognized that blood collected from sick patients separated into four distinct layers. It was thought that these humors which consisted of the dark clots at the bottom of the tube (melancholic), then the oxygenated red cell layer (sanguine), the fibrin clots (phlegmatic), and the supernatant serum (the choleric), were the actual cause of disease. Musgrove,[31] 1703, observed the buffy clot in the blood of rheumatic subjects. John Hunter[21] recognized that the increased sedimentation was associated with the granularity of the blood which was later described by Lister[25] and Hodgkin[26] as rouleaux formation. Fahraeus,[9,10,11] in 1918, was the first to make a study of the sedimentation rate using an anticoagulant and a simple technic to establish the normal limits of the procedure. This application of the sedimentation rate in the study of acute and chronic rheumatism was made by Hermann[20] in 1924. Subsequently, many investigators[1,5,7,8,10,22,24,31,35,36,43,44] have confirmed his observation, *i. e.*, that the rate is a rough index of the intensity of the disease, especially in rheumatoid arthritis and rheumatic fever. (See chart).

In order to interpret the meaning of the sedimentation rate properly, one should take into consideration the factors which influence the rate of fall of th[illegible] red blood cells. [illegible] sedi-
[illegible] [29]
[illegible] is
they form solid masses, there is a greatly accelerated sedimentation. The normal suspension of erythrocytes is dependent upon the hydrophilic property of their surface layers and their negative electrical charges.[23,29,41]

The phenomenon causing rouleaux formation is poorly understood and the sedimentation rate is really an index of the size of these rouleaux masses.[29] This formation is easily disturbed by shaking or by dilution of the plasma with electrolytes such as sodium chloride or citrate. The tendency of the cells to form rouleaux seems to be an attraction rather than

What the Clinician Wants to Know From X-rays of Involved Joints

1 [illegible]

what is the stage of the arthritis?

2 Are the ends of the bones covered with cartilage? The joint space indicates, in general, the thickness of the cartilage. Degenerative *changes in a joint are roughly inversely proportional to the thickness* of the cartilage How much narrowing of the joint space has taken place?

3. Are the articular parts in normal alignment? This is important in determining a plan of reconstruction and in preventing further deformity.

4 What is the condition of the bone on the articulating surfaces and adjacent thereto? What can be said of the zone of provisional calcification? Ground glass atrophy and the width of the joint space can be followed, giving an idea as to progression of disease. Also "punched out" areas should be noted

5 Are there any changes in contour of the bones—especially in such joints as ball and socket and weight-bearing joints—such as flattening of the head of the femur?

6 What are the conditions within the joint space? villi? joint mice? soft tissue swelling?

7. Do abnormalities exist in the periarticular soft tissues? Calcification in bursæ or in ligaments of the spine? Does tenosynovitis exist? Are the swellings concentric or eccentric? Is there subperiosteal hemorrhage? Are bone tumors or fractures present?

8 The internist would like an estimate, if possible, of the general bony texture and structure—the mineral content of the bone. The structure and mineral content of a bone should be determined at a distance from [illegible]

Alterations in calcium content (osteoporosis and osteosclerosis) frequently are evident in arthritic joints. The presence of osteoporosis of the epiphyses with slight narrowing of the interosseous space will suggest rheumatoid arthritis, however, if considerable bony lipping is present, degenerative changes (osteo-arthritis) must also be considered. The osteoporosis frequently seen in the shoulder joint following long immobilization for traumatic periarthritis might suggest an early rheumatoid arthritis When there is definite destruction of bone and cartilage, it is very difficult from the roentgen picture alone to distinguish a gonorrheal arthritis from severe rheumatoid arthritis or even a degenerative hip joint disease (malum coxæ senilis).

Periarticular swelling may occur in acute gout, early gonorrheal arthritis, early rheumatoid arthritis, periarticular fibrositis and rheumatic fever. The roentgenologist cannot distinguish these unless additional changes are present, such as atrophy of bone, loss of articular space and abnormality of bony contour.

a fusion because they can be easily disengaged. The cause for the rouleaux formation is the result of changes in the *plasma* rather than in the cells, themselves. If specimens of blood showing excessive rouleaux formation are washed free of the plasma, they lose this activity if they are again suspended in normal plasma or saline

The addition of human plasma fibrinogen or serum globulin to saline suspensions of red cells causes rouleaux formation. An increase in the concentration of these proteins accelerates the settling velocity of the rouleaux masses[17] The simplicity of the test for determining the sedimentation rate of red cells makes available a semi-quantitative method for estimating the fibrinogen (globulin) content of plasma. An elevated sedimentation rate does not always indicate an increase in the fibrinogen concentration An increase of the serum globulins, as in cirrhosis of the liver, may be accompanied by a marked elevation in the sedimentation rate even though the fibrinogen concentration is normal.

Certain of the abnormal proteins such as gelatin, nucleic acid and hyaluronic acid are all capable of increasing rouleaux formation and thereby causing an elevated sedimentation rate. The property of hyaluronic acid to increase the sedimentation rate is common to all high polymer linear colloids It has been suggested that in rheumatic diseases these

cent solution) Potassium oxylate or sodium citrate should not be added in a crystalline form to the blood since they cause shrinkage and crenation of the red cells and, in this way, interfere with the rouleaux formation. The shrinkage effect produced by potassium oxylate can be counteracted by the addition of ammonium oxylate which tends to increase the red cell size

red cell column, referred to as the "period of free fall," which is determined by the length of the tube The third and last period occurs when the cells begin to pack in the bottom of the tube and is referred to as "period of packing."

Another factor which should be taken into consideration is that the sedimentation tube should be *vertical* in order to prevent the red cells from sliding down along the inclined plane of the wall of the tube. The tube should be at least 2 5 mm. in diameter and the blood specimen should be carefully shaken to break up the rouleaux formation prior to setting up the test.

Hypertrophic changes are usually the dominant ones in degenerative arthritis (osteoarthritis); however, atrophic changes may be modified by weight in weight-bearing joints (such as the ankles) so that these may show more bony hypertrophy than atrophy. In addition, hypertrophic changes may be seen late in rheumatoid arthritis, in gout, and in traumatic joint disease Hypertrophy of bone may be present in the terminal phalangeal joints in psoriatic arthritis Hypertrophic changes are sometimes seen in the spinal column involved by tuberculosis, or in typhoid arthritis of the spine. Degenerative joint disease may closely simulate the picture of the reparative process of various forms of infectious arthritis (tuberculous, gonorrheal, pneumococcal and syphilitic) [44]

Patients diagnosed clinically as "arthritis" may be found by x-ray studies to have these symptoms secondary to myeloma, carcinomatous metastasis to bone, tuberculosis of the kidney, calculi in the urinary tract, Paget's disease (osteitis deformans) etc [110]

RHEUMATOID ARTHRITIS

Ther [illegible]
type [illegible]
spondy [illegible]
Strumpell arthritis), psoriatic, and atypical rheumatoid The roentgen findings in the different clinical forms of rheumatoid arthritis vary with the joints involved, the stage of the disease and the severity of the process.

Adult Rheumatoid.—Lupo's report of the radiology of chronic joint [illegible]

atrophy of disuse, because the former is more homogenous, is confined to spongy tissue and stands out in contrast to the relatively increased density of the cortex (Fig. 8.)

2 Progressive narrowing of the joint spaces, extending uniformly among the whole line of the joint (Figs. 9, 12).

3 Plastic, waxlike deformation of the epiphyses, due to shrinking of the joint capsule.

4. Lastly, destruction of the articular extremity and disappearance of the joint line and bony ankylosis (Figs 9 (5), 10, 16, 19)

Taylor and his co-workers noted systemic decalcification, atrophic bone destruction, joint effusion, narrowing and obliteration of the joint space and evidence of bone production (lipping and osteophytes) in more than 75 per cent of cases of rheumatoid arthritis. Systemic decalcification was most marked in the bones of the hands and feet, although it was noted throughout the skeleton One should always obtain lateral views of the small joints of the hands or one may miss large areas of destruction.[128] Decalcification of bone will aid greatly in identifying the rheumatoid arthritic. As the disease becomes more quiescent, there is increased density of the shafts of the bones. These workers pointed out that punched-out areas of bone are a prominent feature of rheumatoid arthritis and occur as often in this disease as in gout (although such areas are usually somewhat larger in the latter disease). In rheumatoid arthritis, these lesions app

The methods described by Cutler, Linzenmeir, Rourke-Ernstene, Westergren and Wintrobe are all reliable and in common use. The Rourke-Ernstene method is the most accurate method because it uses heparin as the anticoagulant, the rate is estimated from the period of free fall, and the result is corrected by the hematocrit index. This method is time-consuming and for that reason is not a practical test for the general practitioner The Westergren, Wintrobe, Wintrobe-Landsberg (Hynes and Whitby modification),[17,50] and Cutler methods are recommended for use in office practice.

The most practical method is the Westergren technic which is measuring the distance fallen at the end of an hour. The length of the tube used is very important because it must be long enough to take advantage of the long period of free fall and, in this way, the packing of cells in the bottom of the tube can be ignored The 200 mm. tubes[47] are recommended for this method

Interpretation of the Sedimentation Rate

Normal Values	Millimeters per Hour	
Method	*Men*	*Women*
Westergren	0 to 15	0 to 20
Wintrobe	0 to 6 5	0 to 15
Cutler	0 to 8	0 to 10
Rourke-Ernstene	0 05 to 0 4 mm. per minute	

WESTERGREN TECHNIC

Apparatus Required.—Sterile 5 cc syringe, needle, a solution of 3 8 per cent sodium citrate in distilled water, a special Westergren pipette, at least 300 mm long, and calibrated from zero to 200 mm., a special rack to hold the pipettes in a vertical position. The diameter of the Westergren tube should be at least 2 5 mm

Technic.—Enough anticoagulant should be placed in a test tube so that 0.2 cc. of the anticoagulant is available for each 0 8 cc. of whole blood The tourniquet should be removed before withdrawing the blood from the vein The blood is then placed in the test tube with the anticoagulant and inverted several times in order to prevent clotting. The Westergren pipette is then filled to the 200 mm mark and placed in the vertical rack at room temperature At the end of an hour, the reading is taken of the height of the packed red cells and recorded in millimeters fall per hour

Normal Figures.—The normal range for the Westergren technic varies from zero to 15 mm per hour in men, and from 0 to 20 mm. per hour in women.

The presence of anemia, which is frequently encountered in subjects with arthritis, is also another disturbing factor in the determination of an

as though bone were removed with a punch; no reaction is noted in adjacent bone (Figs 9 (3), 38*A*).

Kuhns[78] found the shoulder to be involved in only 9 per cent of 1765 patients with rheumatoid arthritis. Roentgen changes were rarely marked in these individuals and symptoms referable to the shoulder were frequently severe (Fig 14). Involvement of the shoulders was usually bilateral, progressed slowly, usually without the exacerbations which affect the weight bearing joints, and in some cases almost painlessly.

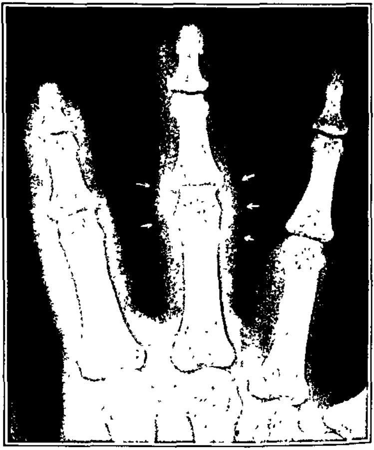


FIG 8 —Rheumatoid arthritis in a woman of thirty-six Arrows point to fusiform periarticular swelling around proximal phalangeal joint of middle finger. This joint space is narrowed Bony trabeculae are hazy This represents a fairly early stage of rheumatoid arthritis

per cent
War II

ntgeno-
cases of rheumatoid arthritis
15 and 17). They noted that
l for long periods following

abnormal sedimentation rate [illegible]
blood from normal people, [illegible]
information, he has been a[illegible]
portional increase in the sedimentation rate to the decrease in the volume of the packed red cells. The criticism of this study is that it does not consider the abnormalities of the blood plasma, *which are responsible for* the increased sedimentation rate. Whitby and Hynes[19] have demonstrated that there is a considerable variation with dilution if one uses plasma obtained from different patients having abnormal sedimentation rates. Their work demonstrates that there are factors in the plasma other than the cell volume which affects the sedimentation rate, which cannot be corrected by estimating the cell volume alone. McFarlane and O'Brien[20] have improved the accuracy of the method by first centrifuging the whole blood and removing the excess plasma, and then re-mixing it in proper proportions before doing the sedimentation rate.

Information derived from the use of the sedimentation of red cells should always be correlated with the clinical findings, should always be regarded as a non-specific test, which is for the most part an index of tissue injury, and in a rough way measures the amount of activity of rheumatic disease or other disease processes causing tissue destruction. For this reason, it can be used as a screening procedure to detect conditions which cause an increase in plasma fibrinogen or serum globulins or both. The *test has been most valuable in following the course of chronic infections,* as rheumatic fever, rheumatoid arthritis, and tuberculosis. The test frequently is valuable in the detection of occult infection, neoplasms, and cirrhosis of the liver.

Sedimentation Rate Obtained in Various Diseases

Joint Diseases		Other Diseases
Normal	Elevated	
Degen. Joint Disease (osteoarth.)	Rheumatic Fever	Pregnancy
Fibrositis	Chorea	General Infections,
Psychogenic Rheumatism	Rheumatoid Arthritis	Menstruation
Traumatic Arthritis	Suppurative Arthritis	Hyperthyroidism
Neurogenic Arthropathy	Gonococcal	Hepatic Disease
Palindromic Rheumatism	Streptococcal	Leukemia
	Other bacteria	Lymphoblastoma
	Tuberculous Arthritis	Anemias
	Multiple Myeloma	Chronic Infections
	Malignancy, Joint	Tuberculosis
	Reiter's Disease	Nephritis (Nephrosis)
	Acute Lupus Erythematosus (disseminated)	Visceral Infarction
	Periarteritis Nodosa	Metastatic Malignancy
	Gouty Arthritis	Rheumatic Carditis

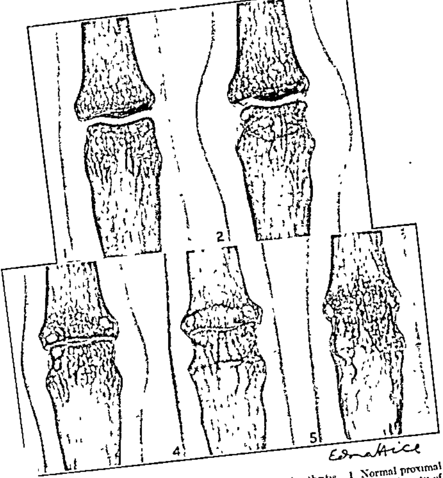


Fig 9 —Roentgen findings in progressive rheumatoid arthritis 1 Normal proximal halangeal joint of finger There is no periarticular swelling Note normal density of one, and absence of cystlike cavities 2 Early rheumatoid arthritis Note the fusiform oft tissue (periarticular) swelling with some haziness of the joint space 3 Further progress of the disease with narrowing of the joint space indicating destruction of cartilage, demineralization of the bone and beginning formation of small bony cysts near the joint margin 4 As the disease advances, further demineralization occurs, with further narrowing and almost complete obliteration of the joint space, with diminution of the soft tissue swelling 5 Late stage The joint space may entirely disappear (indicating complete destruction of cartilage); there is bony ankylosis with some increase in the mineralization of the bone as compared with 4 The soft tissue swelling may disappear as in this instance

THE CLINICAL INTERPRETATION OF THE SEDIMENTATION RATE

Degenerative Joint Disease (Osteoarthritis).—The sedimentation rate in primary degenerative joint disease is usually normal. In some instances, mild elevation in the sedimentation rate may be encountered in subjects

general rule, the sedimentation rate is normal in degenerative joint disease and, for that reason, it is useful in excluding rheumatoid arthritis.

Fibrositis.—The sedimentation rate is normal in the syndrome of periarticular fibrositis which is characterized by morning stiffness and pain which responds to exercise, salicylates and hot applications. Slocumb[illegible] has recently point
the diagnosis
arthritis. Fr
passage of considerable time.

Clinical Application of the Sedimentation Test

In general, the sedimentation rate becomes rapid in the presence of inflammation, tissue damage (as in neoplasm), severe trauma, local and generalized infection, anemia and pregnancy.

Moderate abnormalities accompany leukemia, hyperthyroidism, liver diseases with jaundice, coronary occlusion and thrombosis, nephritis, starvation and acute intoxication

Acute respiratory infection and chronic sinusitis do not increase the sedimentation rate unless they are severe and present over a long period of time

When the sedimentation rate indicates an active process, it gradually returns to normal as the patient improves. For this reason the test

farction, pneumonia and subsiding infections.

The presence of a normal sedimentation rate does not rule out disease and is helpful only when it is significantly elevated.

The sedimentation rate is useful first as a diagnostic aid and, second, as a measure of the severity of disease

In conditions in which there is a large element of psychoneurosis, the presence of a normal sedimentation rate is reassuring to both the physician and the patient

In fibrositis, the finding of a normal sedimentation rate is one of the necessary diagnostic criteria

The sedimentation rate is the best index of the severity and progress in rheumatoid arthritis, rheumatic fever, and specific infectious arthritis

The test is especially of value in the mild, early cases where the physical findings are either absent or atypical

In instances where there is a markedly elevated sedimentation rate associated with skeletal pain which cannot be controlled with salicylates and requires opiates, one should suspect the possibility of malignant disease.

Roentgen Findings in Rheumatoid Arthritis

1. No changes may be apparent very early in the disease.
2. The *first roentgen sign* is usually *periarticular soft tissue swelling*—a fusiform swelling of the soft tissues about the joint, indicated by increased density of the soft tissues (due to proliferation of synovial membrane, joint effusion and thickened joint capsule).[123,124,142]
3. *Generalized skeletal decalcification* is an early sign.[29] The decalcification is usually more marked in the articular ends of the bones This demineralization cannot be distinguished roentgenologically from that of hyperparathyroidism, hyperthyroidism, rickets, osteomalacia or senile osteoporosis. There is *rarefaction of the trabeculated ends of the bones*, followed by atrophy of the ends of the bones, thinning of the articular cortices and a *ground-glass appearance* and atrophy of disuse in the shaft Generalized decalcification of the bony skeleton is one of the most characteristic roentgen findings.
4. Definite *effusion within the joint* presenting a dense, sharply outlined and fusiform appearance.
5. Progressive *narrowing of the joint space* (indicating thinning of the articular cartilage).
6. [illegible]
7. The *articular surfaces become irregular and may be destroyed;* this may be followed in some cases by subluxations and dislocations.
8. Marked *transparency of bones.*
9. Hypertrophy of bone may appear in the late stage; this may be seen relatively early in weight-bearing joints.
10. In the *very late stages* of severe rheumatoid arthritis, very little if any periarticular swelling remains, but there is marked *atrophy of bone*, destruction of cartilage, moderate *bony lipping* and *osteophytes* (in some cases), marked destruction of bone, *disarrangement of the joint and deformity*[21] *Fibrous or bony ankylosis* may occur, resulting in continuity of the trabeculated ends of adjacent bones. Bony ankylosis is most common in joints with a narrow range of motion, such as the sacro-iliac joint or carpus. Surrounding muscles become atrophied and occasionally small calcified deposits may be seen about the joint margins One may see deviation, luxation or subluxation, thickening of the bones and deformity of the bony ends at the joints, due to mechanical forces If the disease becomes quiescent, there may be an increase in bone density. Occasionally, telescoping of the ends of the bones occurs with the development of the opera glass hand (*la main en lorgnette*) [28] (Fig. 34)

Bone Absorption in Rheumatoid Arthritis.—All of the reported cases of the opera-glass hand (la main en lorgnette) have had "polyarthritis" for an average of twenty-four years and each of four additional cases recently reported by Smyth[163] had had rheumatoid arthritis for many years Absorption of the head of the femur resulting in the deformity of protrusio acetabuli (Otto pelvis) occurs in patients with advanced generalized rheumatoid arthritis. The term "polyarthritis mutilans," introduced by Stursberg,[166] has been applied to cases of marked bone absorption of

Gout.—The sedimentation rate is increased during the acute attacks with a fall to normal during the free period Late in the course of gouty arthritis the test may remain high This elevated rate does not parallel the blood uric acid increase.

Rheumatic Fever.—One of the characteristics of acute rheumatic fever [illegible]

rheumatic fever. Electrocardiographic studies made during this period indicate that abnormalities consistent with rheumatic myocarditis may appear before the development of the acute periarticular phase characteris- [illegible]

In the presence of cardiac decompensation, a normal rate may be encountered and, in this instance, it is of no value in indicating the severity of disease The author has recently seen such a patient who died as the result of marked valvular disease, and rheumatic myocarditis proved by necropsy. There is reason to believe that acute rheumatic fever without cardiac failure may exist in the presence of a normal sedimentation rate Such cases are the exception and a diagnosis of rheumatic fever should always be questioned if the rate remains normal.

Chorea, which is considered a manifestation of rheumatic fever, is also characterized by an increase in the sedimentation rate[2] Others have found the rate may be normal in chorea without other manifestations of rheumatic fever[3,5,40]

[illegible]

method, found that the test usually exceeded 30 mm per hour in patients with active disease Short, Dienes and Bauer,[41] using the Rourke-Ernstene method, found that the sedimentation rate was positive in 92 2 per cent of the tests and they concluded that it was much more accurate than the Schilling count (87 per cent). The Vernes' test and the streptococcus agglutination reactions carried out on the same subjects were positive in approximately 50 per cent, respectively.

APPLICATION OF THE SEDIMENTATION RATE IN ARTHRITIS

The use [illegible] the mechanical [illegible] like conditions. [illegible] tive [illegible]

arthritis The sedimentation rate is increased during the acute gouty attacks and returns to normal following the attack.[4 15 16 27 21] There seems to be no relationship between the degree of sedimentation and the uric acid blood level[15]

advanced rheumatoid arthritis and to similar changes occurring in thrombo-

arthritis occurring in a young age group.



FIG. 10 —Advanced rheumatoid arthritis Hand and wrist show a moderate amount of demineralization of the bones There is narrowing of all of the joint spaces of the metacarpals and phalanges Similar changes are present in the carpal bones to a more marked extent Here all the normal architecture of the wrist bones is lost and there is ankylosis present between these bones and the radius and ulna

di·

wi

bones Later the periarticular swelling subsides and atrophy of soft tissues is common Progressive bone decalcification, possibly cortical cysts, may occur As the cartilage is destroyed the joint space is reduced and may be

Rheumatic fever is characterized by excessive increases in the sedimentation rate—frequently in excess of 100 mm. per hour. This abnormality is considered the most sensitive test for rheumatic activity.[5,14,18,23,30,45] Most observers conclude that there is seldom an increase in the sedimentation rate in chorea unless there are other manifestations of rheumatic fever;[3,5,11] however, Bauer[2] reported a group of 70 patients with abnormal rates using the Rourke-Ernstene technic.

The sedimentation rate is very helpful in the diagnosis and treatment of rheumatoid arthritis because it roughly parallels the extent and severity of the arthritic process For this reason it is very useful as a measuring-stick of the effectiveness of various forms of therapy, especially gold therapy. A normal sedimentation rate should be one of the criteria in concluding that a patient has become quiescent or cured of rheumatoid arthritis One of the striking features of rheumatoid arthritis is that the sedimentation rate may remain elevated for years with little fluctuation The presence of an [illegible]

BIBLIOGRAPHY

1 ALSTON, J M: The Practitioner, 157, 23, 1946
2 BAUER, W. New England Jour Med, 221, 524, 1939
3 BENNETT, A E, and HOEKSTRA, C. S Jour. Omaha Mid-West Clin Soc, 2, 59, 1941
4 BRØCHNER-MORTENSEN, KNUD Acta Med Scand, 106, 81, 1941.
5 CLARKE, P L H Irish Jour Med. Sci, 817, 1940.
6 COLLINS, D H, GIBSON, H J, RACE, J, and SALT, H B Ann Rheum Dis, 1, 333, 1939
7 DAWSON, M H, SIA, R H P, and BOOTS, R H. J Lab & Clin Med, 15, 1065, 1930
8 DITGES, H J Deutsche Med Wchnschr, 55, 1171, 1929
9. FAHRAEUS, R Biochem Ztschr, 89, 335, 1918
10 ——— Physiol Rev, 9, 241, 1929
11 ———: Stockholm, P A Nordstedt and Söner, 1921
12 GIBSON, H J The Practitioner, 157, 28, 1946
13. GIBSON, H J, and KERSLEY, G. D Med Pr, 196, 353, 1938
14. GIBSON, STANLEY J Tennessee Med Assoc, 34, 249, 1941
15 HADEN, R L, and KINNELL, G Jour Lab. & Clin Med, 27, 725, 1942
16 HADEN, R L Cleveland Clin Quart, 8, 230, 1941
17 HAM, T. H A Syllabus, *etc*, Harvard Press, Cambridge, 1951.
18 HARRIS, T W Am Jr Med Sci, 210, 173, 1945
19 HENCH, P S, BAUER, W, BOLAND, E W, CRAIN, D C, FREYBERG, R H, GRAHAM, W, HOLBROOK, L, LOCKIE, M, MCEWEN, C, ROSENBERG, E. F., and STECHER, R. M Ninth Rheumatism Rev, Ann Int Med, 28, 318, 1948
20 HERMANN, H: München Med Wchnschr, 71, 1714, 1924
21 HUNTER, JOHN Works ed by Palmer, London, 1, 235, 1835
22. KAHLMETER, G Klin Wchnschr, 5, 889, 1926
23 [illegible] Jour Pediat, 18, 337, 1941
24 [illegible]
25 [illegible]
26 [illegible]
27 [illegible]
28 MCCRACKEN, J H Bull New Eng Med Center, 3, 249, 1941.
29 MCFARLANE, R G, and O'BRIEN, B. M The Practitioner, 157, 17, 1940

obliterated in severe cases by bony union. As the disease progresses subluxations and deformities are frequently noted. Caffey[20] describes two roentgen changes in juvenile rheumatoid arthritis which are not observed in the adult disease. The first is regional acceleration of maturation in the bones which subtend the inflamed joints accounting for the brachydactylia found in long-standing cases, the second is regional external cortical thickening usually most conspicuous in the small tubular bones of the hands and feet.

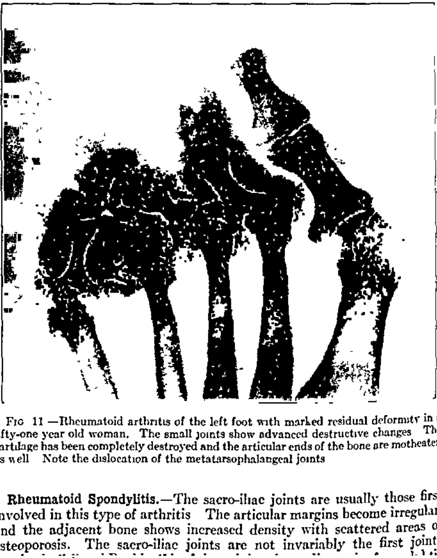
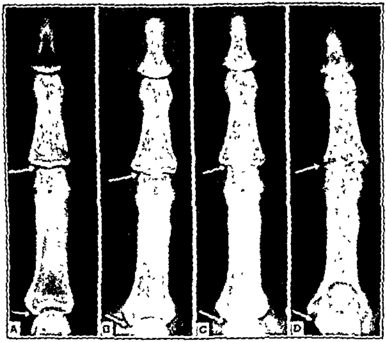


FIG 11 —Rheumatoid arthritis of the left foot with marked residual deformity in a fifty-one year old woman. The small joints show advanced destructive changes The cartilage has been completely destroyed and the articular ends of the bone are motheaten as well Note the dislocation of the metatarsophalangeal joints

Rheumatoid Spondylitis.—The sacro-iliac joints are usually those first involved in this type of arthritis The articular margins become irregular, and the adjacent bone shows increased density with scattered areas of osteoporosis. The sacro-iliac joints are not invariably the first joints [illegible]

joint include narrowing of the joint space; finally ankylosis with marked osteoporosis of the adjacent bone.



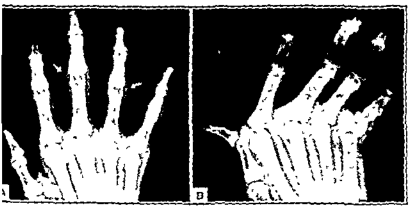


Fig. 13.—The right hand (A) of a 40 year old female showing soft tissue swelling of the proximal phalangeal joints, A sign of early rheumatoid arthritis. Three years later (B) the same hand shows late changes with ulnar deformities and subluxations of the metacarpo-phalangeal joints.

an obese man with chronic rheumatoid arthritis will often have typical

Moderately advanced gonorrheal arthritis may show osteophytes and areas

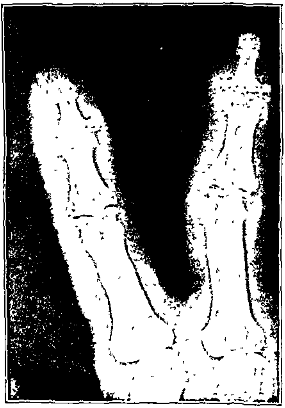


Fig 21 —*Degenerative joint disease* with formation of *Heberden's nodes* at terminal phalangeal joints Note also some involvement of proximal phalangeal joint (which

of osteosclerosis in the subcortical bone and therefore have in the same joint features of two types of joint disease The term *mixed arthritis* should be *qualified* or not used (*e g*, rheumatoid arthritis, severe, osteoarthritis, mild)

ARTHRITIS DUE TO SPECIFIC INFECTION

Tuberculous Arthritis.—Roentgen findings may be absent in the early stages of this disease. There are no findings which are entirely typical of

The apophyseal joints of the spine are frequently affected; the roentgen criteria of involvement of these joints are deossification of articular proc- [illegible]

spondylitis by Oppenheimer,[93] but not by Boland and Present[11] who, even when special techniques were used, found lesions in the apophyseal joints less definite and constant than those in sacroiliac joints. In none of their cases were changes in apophyseal joints found without accompanying involvement of the sacro-iliac articulations. When apophyseal changes were found, usually only a few scattered apophyseal joints were affected while intervening joints were not involved.



FIG 14.—Anterior-posterior view of the left shoulder of a 44 year old woman with advanced rheumatoid arthritis The joint margins are irregular and the glenoid fossa is in contact with the head of the humerus There is demineralization of all bones of the shoulder joint

This disease frequently produces calcification of the anterior and lateral vertebral ligaments (Figs. 20 and 21) Oppenheimer reported this in 50 per cent of his cases Only a few vertebrae may be united by a thin calcium encasement, however, extensive involvement with ossification occurs in advanced cases producing the so-called "bamboo spine." [illegible]

course of the disease hip and shoulder joints and large or small peripheral oints may also become affected and in such cases the roentgen changes are similar to those in typical adult rheumatoid arthritis.

tuberculosis of joints in any stage.[55] Although the findings vary greatly with the stage and intensity of the process, the usual roentgen signs include:[38] synovitis, periarticular swelling, atrophy or sclerosis of bone, production and destruction of bone in the epiphysis and metaphysis, destruction of cartilage as seen by narrowing of the joint space, sequestration and the finding of cold abscesses or sinuses. Sequestra never form in other types of arthritis.



FIG 25 —Hypertrophic changes in the lumbar vertebræ. (Holmes and Robbins)

The joints most commonly affected are the hips, spine, knees, shoulders, wrists (Figs. 28 and 29B), and elbows; the small joints of the hands and feet are rarely affected in the adult. Involvement is usually mono-articular with evidence of tuberculosis elsewhere (lungs, kidneys). Most cases occur in children under fourteen years of age; it occasionally occurs in adults.



FIG. 15.—Rheumatoid arthritis of the ankle joint in a white male age thirty years. Lateral views of the left foot taken three and one-half years apart. The tibio-tarsal and intertarsal joint spaces in the lower roentgenogram are hazy or obliterated, spur formations along the joint margins have developed. The bones of the foot have become more dense.

Pomeranz advised that one suspect tuberculosis in the presence of the following: wedge or cone-shaped lesions, "kissing" sequestra; narrowing of a joint space occurring late in an infection, despite associated destructive changes; complications such as kyphosis, sinus formation and cold abscesses. Bone atrophy usually develops before destruction develops This distinguishes tuberculosis from pyogenic types of arthritis.



FIG. 26.—*Degenerative joint disease* seen here involved the *thoracic vertebræ* (in a patient aged sixty-seven years). There is *marked lipping with complete bridging* across the intervertebral spaces of some of the thoracic vertebræ The appearance resembles that of a *flowing hyperostosis* These changes represent an *advanced stage of degenerative joint disease*

Roentgenograms of the spine may fail to reveal spinal tuberculosis; frequently the full damage is not revealed in roentgenograms taken in the anteroposterior view and no attempt at diagnosis should be made without good lateral roentgenograms. The earliest change is a slight thinning of the disk; this progresses to produce destructive changes involving the adjacent surfaces of the two vertebral bodies; finally there is complete

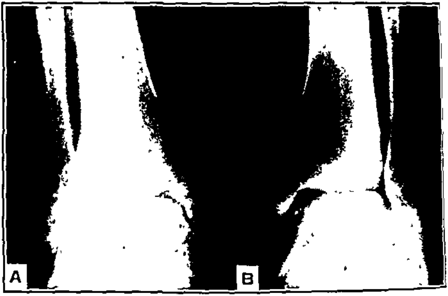


FIG 16 —The ankles of a negro man age 50 years with advanced rheumatoid arthritis on the right (A), the opposite ankle (B) shows a normal joint space

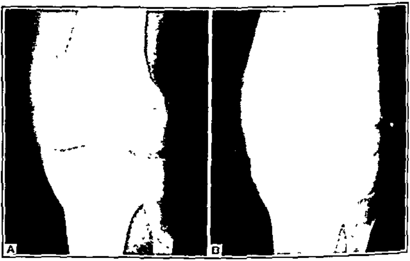


FIG. 17.—Rheumatoid arthritis in the left knee The first roentgenogram (A) was taken ten years before the second (B) There has been loss of articular cartilage (reduced joint space), sclerosis and marginal proliferation of bone

disappearance of the disk, advanced caries and collapse of the vertebral bodies. The healing phase is characterized by new bone formation which may result in the production of a single pyramidal shaped mass of bone. Bridging of the vertebrae, formerly considered indicative of secondary infection, is probably a reaction of the periosteum with new bone formation or ligamentous calcification.

Roentgen Differentiation of Rheumatoid and Osteo-arthritis

	Rheumatoid Arthritis	Osteo-arthritis
Earliest signs	Fusiform swelling of soft tissues and rarefaction of trabeculated ends of bones	Formation of osteophytes and narrowing of the joint space
Soft tissue involvement	Fusiform swelling about joints, especially in phalanges	Usually none
Lipping and osteophytes	In about 75 per cent of cases	In every case
Heberden's nodes	None	Very frequent
Punched-out areas of bone at articular margins	Very common	Usually absent
Systemic decalcification	In almost every case, may be the earliest roentgen sign	Mild and consistent with age of patient
Narrowing of joint space	In almost every case	In about 50 per cent of cases
Ankylosis (fibrous and bony)	In approximately 25 per cent of cases	Very rare, only in spine

Roentgen Differentiation of Gout and Rheumatoid Arthritis

	Rheumatoid Arthritis	Gout
Punched-out areas of bone at articular margins	Common (over 80 per cent of cases)	Equally common; areas usually larger
Systemic decalcification	Common	*Absent* except to mild degree consistent with age of patient
Thinning of articular cartilage	In almost every case	In about two-thirds of cases
Periarticular swelling	Symmetrical, fusiform	Asymmetrical swelling about joints
Bony ankylosis	In about 25 per cent of cases	Very rare
Involvement of metatarsophalangeal joint of great toe	May occur—not characteristic	Very frequently involved
Joints involved	Proximal interphalangeal joints of hands, metacarpo-phalangeal joints, wrists, knees, spine; others less often	Especially hands and feet, may affect any joint but is rare in spine, hips and shoulders

usually symmetrical and is distributed along the margins of the joints. He finds the earliest evidence of joint disease, as distinct from bone, in the posterior articulating facets of the vertebrae shown by rarefaction of the articulating processes and blurring of the facet outlines. He contends that the stage of parasacroiliac osteosclerosis passes early into ankylosis of the



FIG. 18.—Close-up of moderately advanced rheumatoid arthritis of the fingers in a woman aged twenty-five years. The soft tissue swelling is evident at the proximal phalangeal joints, together with the narrowed joint spaces and ground glass appearance of some of the bones.

In early tuberculosis of the hip and in septic hips the intrapelvic shadow

should lead one to suspect tuberculous arthritis. The joint damage in tuberculous arthritis requires much more time (months or years) for its development than in other forms of pyogenic arthritis (days or weeks)



FIG. 27.—Mixed form of arthritis in a woman aged seventy-four years X-ray of the knees shows considerable hypertrophic and some atrophic changes, this began shortly after the menopause (at the age of fifty)

Tuberculous arthritis in old age may resemble hypertrophic or septic arthritis. Punched out areas in the shaft immediately adjacent to the involved joint are considered to be almost characteristic of tuberculosis of the shoulder, knee, ankle and especially of the elbow joint.[91]

Tuberculosis of the sacro-iliac joint is a relatively rare condition and usually involves only one sacro-iliac joint In a recent study[155] of 23 patients with known tuberculosis selective obliteration of one joint was

Roentgen Findings in Rheumatoid Spondylitis

1. Changes in the sacro-iliac joints (narrowing or obliteration of the joint

2. *Narrowing of the apophyseal joint spaces.*
3 Irregularity of the outlines of the facets.
4 Bony ankylosis of the articulations.[45,114]
5

6.

density may be regained after bony ankylosis has occurred

7. No involvement of the intervertebral disks; the intervertebral spaces are of normal width.
8 The ligamenta flava and *longitudinal ligaments* may become *calcified or ossified.*[9]



Fig. 19.—Advanced rheumatoid arthritis in a woman of thirty-nine whose rheumat arthritis began at the age of eleven. Involvement has progressed to the stage of bo ankylosis in the elbow. Marked generalized demineralization is present

reported, in six of these spontaneous fusion occurred while they were under hospital observation.

Syphilis.—Involvement of the joints in *congenital syphilis* is rare and of the gummatous type [129] In the roentgenogram these gummas appear at first as spotty translucencies, later they cause destruction of cartilage and complete obliteration of the joint.

[illegible]

change in such joints is soft tissue enlargement and haziness. Gummatous deposits may occur in the cancellous end of the bone and later spread to

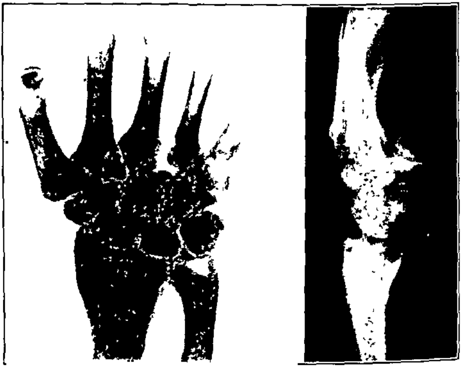


Fig 28 —Tuberculosis of the wrist Anterior and lateral projections of the right wrist in a patient with tuberculous arthritis The intercarpal and carpo-metacarpal joints are indistinct or absent Multiple areas of sclerosis and porosis are clearly seen

cartilage [43,85] One may see erosion and irregularity of the joint surfaces, cavitation within the bones and bony outgrowths. In adults, one large joint is usually involved and the roentgen appearance may resemble degenerative joint disease When the spine is affected, the picture simulates that of tuberculosis. Gumma of the patella has been reported [9] If specific therapy is not instituted, the joint may become ankylosed.

Charcot Joints.—Mitchell first referred to tabetic arthropothies in 1831.[87] Charcot described these joints in 1868 [25] Charcot joints may occur in tabes dorsalis, syringomyelia, and occasionally in leprosy, psoriasis, poliomyelitis, compression and destruction of areas of the spinal cord or following peri-

sacro-iliac joints and is followed by decalcification around these joints as elsewhere in the pelvis and spine

Williams[164] reports that the costotransverse and costovertebral joints are involved in the majority of cases and with adequate *x*-ray examination the earliest evidence of joint involvement can be demonstrated. The technique which he uses to visualize these joints requires an anterior-posterior projection, with the central ray directed 20 degrees cephalad and

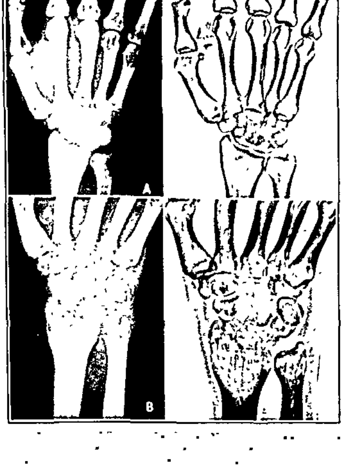


[illegible]

centered between the fifth and seventh thoracic vertebrae. According to this author, advanced changes were found, particularly in females, in these small joints when the sacro-iliac joints appeared to be normal. Failure to demonstrate the small joint changes by roentgen examination may account, at least in part, for the reports of low incidence in the female, and the failure to make the correct diagnosis in the male when there are

pheral nerve section.[133] Usually one major joint is affected but multiple joints may be involved. Charcot joints have been reported in the hip, knees, shoulder, elbow, foot, ankle, spine and rarely in the sacro-iliac joints.[135]

The disease usually begins in the cartilage and the zone of calcification between the cartilage and the underlying cancellous bone.[88] The earliest



changes noted in the roentgenogram are erosion of cartilage, and marginal proliferation of bone. Later one may see chip and compression fractures and ossification in muscles. In the hips, there is early erosion of the acetabulum with irregularity of the joint line. Later, one sees enlargement

the head of the tibia or the condyles of the femur. Pointing of the tibial spines may be an early roentgen change

The hands are often involved and the earliest roentgen evidence of degenerative changes is narrowing of the joint spaces between the terminal phalanges and lateral displacement of the distal phalanx. In the hands these changes occur on the dorsum of the fingers and are called *Heberden's*

marked soft tissue swelling occurs before bony changes are revealed.

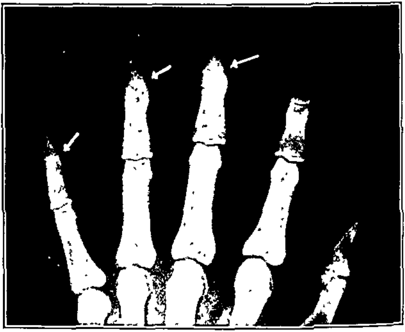


FIG 22.—Psoriatic arthritis. The involvement of the terminal phalangeal joints is not uncommon in rheumatoid arthritis associated with psoriasis. The arrows indicate joint changes in third, fourth and fifth fingers in a female patient with long-standing psoriasis

The earliest roentgenographic changes produced by degenerative joint disease of the fingers is a narrowing of the joint space indicating loss of cartilage. This is followed by thickening of the subchondral bone plate, spur formation, and misalignment of the terminal phalanx. Finally there is broadening of the base of the phalanx with increased density of bone Stecher and Hauser[163] in a study of roentgenograms and case histories of over 100 patients with Heberden's nodes, observed that even in advanced cases uniform involvement of all fingers is rare, individual joints being only mildly altered or even spared entirely. These observers stressed the frequency of proximal interphalangeal involvement associated with degenerative joint disease of the terminal finger joints Such an association was noted in 40 per cent of their cases.

In the foot, one may occasionally note the classical roentgen changes of osteoarthritis in the ankle, dorsal intertarsal and first tarsometatarsal

and destruction of the acetabulum and absorption of the neck and head of the femur. In the knee, roentgenograms reveal early breaking down, condensation and flattening of subchondral bone in the condyles; as the process continues, genu varum or valgum appears and dislocation may occur (Fig. 31). Free bodies are found in the joint in about half of the cases. In more advanced cases, large exostoses are common. Rapid destruction may occur in the short interval of one year,[140] the disease process may gradually progress for years until complete disintegration of the joint has occurred



FIG. 30 —Charcot joint of the left wrist due to syringomyelia in a seventy-six year old man Note the complete dissolution of the carpal bones, sclerosis of the radius and ulna with periosteal reaction. There is an increase in the soft tissue shadow of the wrist with diffuse calcium deposits

Charcot joints occur in 25 per cent of patients with syringomyelia; in this disease the upper extremities are much more frequently affected than in tabes dorsalis (Fig. 30).

Brucellosis.—Spondylitis probably is the most common articular or osseous complication of brucellosis.[96] Sixty-eight cases of brucella spondylitis, usually lumbosacral, have been recorded in the literature.[125] The lesion usually begins in the body of the vertebra and the intervertebral disks are involved secondarily The lesion of brucellosis must be distinguished from a tuberculous process, and also osteomyelitis due to a pyogenic bacteria.

articulations. Similar changes may be demonstrated in acromioclavicular and less commonly in the shoulder joints. *In professional baseball pitchers,* osseous deposits often develop on the posterior inferior border of the glenoid fossa.

Roentgen Findings in Degenerative Joint Disease (Osteo- or Hypertrophic Arthritis)[22,42,124]

Early stage:

Narrowing of the joint space (due to thinning of the joint cartilage)

into the other).

Atrophy of disuse at the ends of the bones in some cases (usually with preservation of the bony trabecular pattern—as differentiated from the ground glass atrophy usually seen in rheumatoid arthritis).

Later stage:

Increase in the size of the osteophytes.

Further narrowing of the joint space which may appear jagged or saw-toothed or almost obliterated

Subluxations of joints may occur

Formation of *cysts at the ends of the bones* and punched-out areas in the borders of the bones. (Fig 38C)

Increase in circumference of the joint due to new bone formation

Condensation of subchondral bone.

Advanced stage:

Osteophytes may appear quite large and irregular

Deformities and deviation of the bones from pressure and traction

between adjacent vertebrae)

Joint mice in some cases.

Joints most affected distal interphalangeal joints of fingers, lumbar and cervical spine, knees, shoulder joint rarely involved.[18]

Joints which are the site of repeated trauma may in time develop excess bone along the margins of the cartilage with the eventual production of secondary traumatic (osteo-) arthritis The *temporomandibular joints* are

show irregular n

mid-life there

compression of

the two affected vertebrae.



FIG. 31.—Charcot joint due to tabes dorsalis. Anterior and lateral views of the left knee of a male patient showing reduction of interosseous space, marked marginal proliferation of bone, erosion of the median table of the tibia and marked sclerosis of the tibia and femur.

Gonorrheal Arthritis.—There are no pathognomonic roentgen signs of gonorrheal arthritis.[89] Suggestive changes include a spotty ground-glass atrophy of the neighboring bones, early acute localized atrophy, bony

tion of osteophytes occur in less than 20 per cent of cases. Narrowing of the joint space, fibrous or bony ankylosis and soft tissue changes are commonly encountered (Fig. 29.1). Gonorrheal arthritis may simulate rheumatoid arthritis, but the roentgen changes are usually more marked and appear within a much shorter period of time.[114]

biconcavity of configuration; possibly with intercentral erosion into cancellous structure (Schmorl's Nodes). In advanced cases ankylosis results from fusion of osteophytes by synostosis. Kyphosis may result from wedge-shaped deformities of a series of vertebrae. The apophyseal joints may present eburnations, irregularities of the facets, marginal spurs and reduction or obliteration of joint spacing.



FIG 23 —Malum coxae senilis Left hip of a man age sixty-six years tion of joint space at the point of maximum pressure of the femoral acetabulum with flattening of the head of the femur

"MIXED ARTHRITIS"

Lupo has described what he considered to be a characteristic roentgen sign of early gonorrheal arthritis which consists of the appearance of a thin line of increased transparency closely beneath the thin line of increased opacity that limits the bone toward the joint cavity. The length of this line is variable and it may extend along the whole joint border. This corresponds to the laminar sequestration which he describes as characteristic of the pathology of gonorrheal arthritis. This sign is less evident in advanced cases

Spur formation on the os calcis was formerly considered to be the result of gonorrheal infection Hench[53] thinks that this is unlikely Gonorrheal spurs are uncommon, having been seen in less than 10 per cent of cases of gonorrheal arthritis Periostitis of the os calcis occurred in 6 per cent of Harkness' 336 cases of gonorrheal arthritis.[49]

Pyogenic Arthritis (Roentgen Aspects) (Pneumococci, Streptococci, Staphylococci, Gonococci)
1. Small, more or less circular *zones of atrophy*—sometimes confluent in polycyclical areas of decreased opacity These areas are in an epiphysis or in the spongy bone. The lesions are sometimes surrounded by a thin halo of diminished transparency.
2 [illegible]
3. [illegible]
4 [illegible]
5. [illegible]
6 [illegible]
7 Ankylosis, as a terminal feature.

ARTHRITIS DUE TO MYCOTIC DISEASE

Actinomycosis.—In vertebral disease a paravertebral phlegmon develops which may produce an abdominal lesion resembling tuberculous psoas abscess. Vertebral actinomycosis is rare (only about 50 cases have been [illegible] necropsy. [illegible] ic" areas [illegible] sclerosis[56] Despite extensive destruction, vertebral collapse is rare; even then intervertebral disks are spared Three cases were reported by Lubert.[80]

Histoplasmosis.—Presumably the first case in which joints were involved was noted by Key and Large[73] Few reports include a description of the roentgen appearance of the joints involved

Coccidioidomycosis.—In 79 cases of 256 collected cases, coccidioidal arthritis has been described as having roentgenograms resembling tuberculous arthritis. In the chronic granulomatous phase the joints most frequently affected were the ankles, knees and feet[107] The locations and nature of the osseous lesions in more than 100 collected cases were discussed[7]

MISCELLANEOUS CONDITIONS

Sarcoidosis (Boeck's Sarcoid) —This is a generalized disease that may involve bone adjacent to joints; although arthralgia may occur, usually the joints themselves are not affected. The bones most frequently showing changes are the phalanges, metacarpals and metatarsals [63] The most common osseous lesions are areas of reticulation with thickening of tra-

ular

ibed

the

and

contain little about the bone and joint lesions. Holt[134] analyzed 37 cases having generalized sarcoidosis with histologic confirmation of the diagnosis and satisfactory roentgen examination of the hands and feet. Of this

15 per cent, had osseous foci These authors also describe in detail the roentgenologic manifestations of osseous sarcoidosis and stress that the diffuse, reticular type of bone destruction resulting in a lace-like appearance is more common than the widely publicized circumscribed foci or punched out lesions at the end of bones Joint involvement in this disease is unusual, occasional examples have been described in children, but they must be regarded as exceptional rarities.

Acromegalic Arthritis.—Erdheim[16] in 1931 described a specific form of

grams of the vertebrae

Widening of the acromioclavicular joint space may or may not occur These changes are of aid in the differential diagnosis of hyperparathyroidism from senile osteoporosis, multiple myeloma and polyostatic fibrous dysplasia

Ochronosis (Alkaptonuria).—The roentgenographic features are unusual

CALCIFICATIONS

Calcareous Deposits about Shoulder.—Calcium deposits about the shoulder joint were found in 2 7 per cent of 6,061 unselected persons studied by Bosworth[14] These deposits are generally considered primary in any of the five tendons of the shoulder,[27 61] which may rupture into the subdeltoid bursa; almost always it involves the supraspinatus tendon which forms the



FIG 41 —Calcinosis of joint capsules in both thumbs in a fifty year old female with schroderma Note the osteoporosis of the adjacent bones

floor of this bursa[60 62 86] In the typical case an amorphous calcification is revealed in the angle between the humoral head, greater tuberosity and acromion The number, size, shape, density and localization of the calcium deposits vary considerably. The mass is generally roughly elliptical (Fig. 43) In the supraspinatus calcifications, they are located just above the junction of the greater tuberosity and head, those of the infraspinatus and teres minor are lower and posteriorly. Baird[1] considered it impossible in most instances to localize these deposits roentgenographically

Multiple deposits occurred in 20 per cent of the 202 affected shoulders reported by Bosworth,[14] 51 per cent of these deposits affected the supra-

hips and shoulder girdle In many of the cases reported there was a well developed osteoarthritis characterized by the production of large osteophytes. The most characteristic roentgenographic findings of alkaptonuric ochronosis are noted in the spine (Fig. 32), pelvis, shoulder and knee joints In the spine the characteristic change is the universal calcification of the *intervertebral disks These appear as elliptical opaque wafers in all of the* intervertebral joints and are most clearly seen in the lumbar area In the shoulders there is always a marked narrowing of the joint spaces so that very little, if any, of the joint is visible. The articular surfaces of the glenoid fossae appear eburnated and fairly large spurs project from their

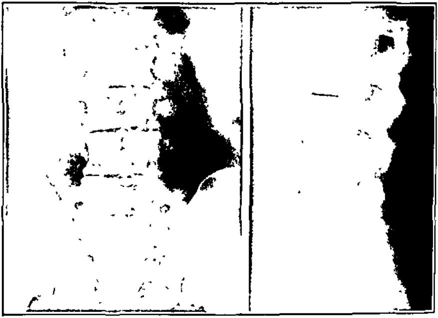


FIG 32 —Anteroposterior and lateral views of the lumbar spine in a male patient age thirty-eight years with ochronotic arthritis admitted because of low back pain The intervertebral disks are thin and calcified, hypertrophic spurs are prominent

margins The humeral heads are somewhat mushroomed and rough circular areas of reabsorption are noted on their articular surfaces. Irregular flocculent calcific deposits are seen in the soft tissues In some cases the pubic cartilage is calcified with fusion of the pubic bones.[99]

Pellegrini-Stieda Syndrome.—In this syndrome there is para-articular calcification at the mesial aspect of the knee joint. The area of increased density lies parallel to the medial epicondyle of the femur and is separated from it by a clear space (Fig. 33) It may, however, be attached to the bone by a pedicle or even fused to the bone Some authors[18,39,90] insist that

'he

ho

a-

Nachlas[87] in a report of 20 cases in soldiers says that in all of these patients there was evidence of arthritis (type not mentioned) in the adjacent

spinatus tendon; 44 per cent had deposits in the infraspinatus tendon and 23 per cent in the teres minor. Horworth studied 100 patients[60] and also found most of these deposits located in the supraspinatus and a few in the other tendons of this joint.

Most writers are of the opinion subdeltoid and subacromial calcific bursitis is never a primary entity but always secondary to some lesion of near-by tendons, generally tendinitis, tear or calcium deposits.[45]

Calcinosis (Chalk Gout).—Calcinosis is a slowly progressing condition of unknown etiology first described in 1878 by H. Weber. Two types are defined, calcinosis circumscripta and calcinosis universalis. In the localized form swellings over joints which occasionally simulate gouty tophi are usually confined almost exclusively to the toes, fingers, elbows and knees.[40,111] These concretions are composed of calcium phosphate and calcium carbonate Multiple disseminated calcinosis may invade skin, form plaques deep in its layers and involve underlying muscles

The association of calcinosis with scleroderma (Fig. 41), acrosclerosis and dermatomyositis has been frequently recorded.[55,141]

ROENTGEN THERAPY IN RHEUMATIC DISEASES

There has been in recent years an increasing interest in roentgen therapy in rheumatic diseases. Carefully controlled studies using this method in

subdeltoid bursitis with or without calcification). Reports of its use in other rheumatic disorders by some observers[13,70,97,135] indicate satisfactory

History.—Soon after the therapeutic employment of roentgen rays reports of their beneficial effect on diseases of the joints began to appear In 1898 Sokolow[122] reported improvement of four patients he had treated with roentgen radiation. In the same year Stenbeck treated 52 patients most of whom he said were "improved." In these earliest reports the type of rheumatic disease was not recorded. Generally good results from treatment of 400 patients with "arthritis deformans" by Staunig[126] in 1925 served to create interest in this form of therapy. Reports by many investi-

of rheumatic diseases. In many cases of acute arthritis (gonococcic, gouty

tated

d) he

t was

recent

natoid

results

joint All roentgenograms showed bony lipping which these authors consider an indication that joint inflammation had been present for some time prior to the precipitating injury.

La Main en Lorgnette (Opera Glass Hand).—This is a rare condition characterized by short stubby fingers with extensive osteoporosis, resorption and partial dissolution of joints of fingers, wrists, elbows, hips with a telescopic type of subluxation (Fig 34). Because joints other than the hands were affected, Crain[33] suggested the term "generalized absorptive arthritis."

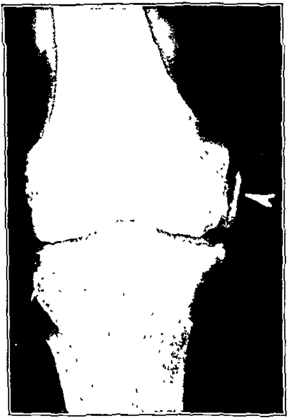


FIG 34 —Pellegrini-Stieda syndrome. Para-articular calcification at the mesial aspect of the knee joint The calcific shadow is not connected to bone in this case

The most striking differential point in this case is the destruction and absorption of cartilage and epiphyseal bone around various joints without ankylosis, resulting in a telescoping of one bone upon the next.

Loose Bodies in Knee Joint.—Loose bodies in the knee joint may result from osteochondritis dissecans, chondrification of the synovial membrane with loose body formation, fracture of large osteophytic processes, or fractures of the articulating surface of femur, tibia or patella (Fig 35). X-ray examinations reveal variability of the position of the loose body.

he obtained with "wide-field" superficial irradiation. He used a special

indicated excellent results, both subjectively and objectively. There is increasing evidence to indicate that roentgen therapy is the treatment of choice in patients with active rheumatoid spondylitis Reports by Foster

follow-up of a little more than seven and one-half years Sixty per cent of these patients indicated that they felt better, 25 per cent felt worse; 15 per cent were the same. Objectively, 5 per cent had complete relief; 28 per cent had no evidence of progression of the disease demonstrable by

of

per

83 cases of ankylosing

intervals, 74 were reli

technique of therapy

2 years, but in this short period, of 60 patients who showed improvement, 14 had subsequently relapsed An analysis of 125 cases treated at the University of California Hospital indicated that any case of rheumatoid spondylitis warrants roentgen therapy, regardless of the duration and severity of symptoms or stage of the disease as indicated by roentgeno-

ective and

therapy to

in 14 per

cent of patients treated with three courses, in 37 per cent receiving two courses and in 55 per cent receiving only a single course

In an experimental study of

52 patients with typical rheum

roentgen radiation over the spi

of roentgen therapy was employed

Physical Factors.—Two hundred kilovolts (175 kilovolts constant potential equivalent) was used, with 0 5 mm of copper and 1 mm of aluminum filtration, a half value layer of 0.9 mm copper, a 50 cm. skin-target distance and an output of 50 r (measured in air) per minute, with the usual size of the field approximately 200 to 300 square centimeters

Dose.—Each field was treated three times, receiving 200 roentgens at each sitting; the treatments were given every other day; the total dose was thus 600 roentgens. Most patients were given three series of treatments to the same ports each series being separated by four to six weeks. All or any portion of the spine (cervical, dorsal, lumbar or sacral) and the sacro-iliac joint, or any combination of these regions, were irradiated. In several

Spondylarthritis.—Spondylarthritis is the term applied by Saenger[18] to a syndrome seen most commonly in children. It is characterized by the roentgenological picture of narrowing of the intervertebral space and evidence of mild destruction of the adjacent portions of involved vertebræ. The changes are slowly progressive for one to two months and then evidence of healing for two to eight months with relatively complete restoration to a normal appearance. It is important to distinguish this syndrome from tuberculous spondylitis. This differentiation must be made from a study of serial roentgenograms, the restoration to near normal appearance within one year occurs in this syndrome, whereas in tuberculosis similar changes occur at best only over a period of two or three years.

HEMOPHILIC ARTHRITIS

In the early stages of acute hemarthrosis, the roentgenograms reveal the distended capsule, widened joint space and shadow of blood (which is denser than synovial fluid). Bleeding into the joint space may be completely absorbed after a few days and the distention of the articular space disappears without residual deformity. Incomplete resorption of blood from the joint space from a single or repeated hemorrhage results in damage to articular tissues with narrowing and irregularity of the joint, deformities and sometimes ankylosis. The epiphyseal cartilages contiguous to a hemarthritic joint shows accelerated maturation and hypertrophy.

The acute hemophilic hemarthrosis may be confused roentgenologically with any traumatic serous or purulent synovitis with distention of the joint space by fluid. The joint is usually partly flexed as the capsule has the greatest capacity (w
entiate these condit
soft tissue clouding
capsule.

In chronic hemophilic arthritis, the joint space becomes narrowed and one sees irregular indented articular bony surfaces, deformity of the ends of the bones, bony cysts, increased density of synovial tissues and a map-like destruction of cartilage (Figs. 36*A*, *B* and 37). In some cases there is atrophy of the bones. Hemophilic arthritis presents no diagnostic roentgenologic picture in the chronic stage and may resemble gout, hypertrophic or tuberculous arthritis.[21]

McDonald and Lozner[81] studied the roentgenograms of joints of 15 adults with hemophilia. In every patient they observed roentgenographic changes in one or both knees, and in all but one patient, in one or both

femoral articular surfaces, and cystic degeneration within the femoral and tibial condyles.

the findings in hemo-

cases of far-advanced disease, involved shoulder and hip joints were also treated.

Considering all patients irrespective of the extent of involvement, duration of symptoms or activity of the disease, 37 (72 per cent) of the 52

[illegible]

sustained subjective and objective improvement. In the late stages, even after calcification of spinal ligaments, significant reduction in pain and stiffness occurred in a high percentage. In 41 per cent of cases, there was a significant decrease in the erythrocyte sedimentation rate.

In order to study the psychic effect that might be associated with roentgen therapy, some patients were treated exactly as described above, but with a lead screen placed to protect the part from irradiation. Care was taken that the patient did not know that such screened parts were not actually treated None of the patients protected from irradiation by lead screens showed any improvement, but without exception, when they were treated first with irradiation directed to one area of the spine, improvement was noted only in the area treated, later a different area of the spine was "treated" using a lead screen, with almost uniformly no beneficial effect.

In patients with early disease (rheumatoid spondylitis), striking relief of pain occurred soon after the first series of treatments, with continued gradual improvement to the point of complete disappearance of all clinical evidence of the disease. When pain in the back was relieved, some patients *with partial calcification of ligaments showed a significant increase* in motion and chest expansion. The beneficial effects persisted in most patients throughout the period of observation. In those whose symptoms recurred several months after therapy, relief was usually obtained by additional treatments. Most individuals reported some improvement within a week after the onset of therapy and some individuals insisted that pain and stiffness were decidedly reduced a few hours after therapy. In others, improvement was less and slower and no change was evident until after the second series of treatments. If improvement did not occur by the time of the completion of the second series, it was unlikely that benefit would follow additional radiation.

Anatomic improvement did not occur following symptomatic benefit from roentgen therapy. Roentgenograms made from six months to two years *after therapy showed no evidence of anatomic improvement*. In some there was an increase in the calcification of the paravertebral ligaments despite significant clinical improvement and reduction in sedimentation rate.

Using this same technique, Robinson[106] has recently reported from this same clinic the results of a long term study of 55 patients followed for 4 5 to 9 years after initial roentgen therapy. Some of the patients were [illegible] in this study can be considered

[illegible]

involved.

emission,

one *for eight years, another for five years.* Seven had recurrences and

Hemophilic arthritis can be differentiated roentgenologically from rheumatoid arthritis by (1) the common involvement of the proximal

arthritis[84]

Subarticular cystic absorption may be accentuated by decalcification of bone by disuse These cysts may vary in diameter from a millimeter to more than a centimeter; they may be single or loculated and round or irregular. Key[72] believed that intra-osseous hemorrhage occurred in bone

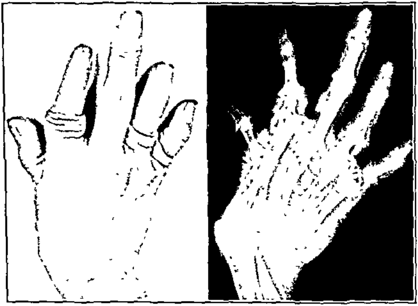


FIG 34.—Opera-glass hand (La Main en Lorgnette) Right hand of a thirty-two year old woman showing a short thumb, index, ring and little fingers with wrinkled skin over the joints The roentgen changes include extensive osteoporosis, resorption of carpal bones and distal ends of the radius and ulna The head and most of the shaft of the proximal phalanges (2, 4 and 5) are resorbed with an evenly rounded proximal end The head of the first metacarpal shows partial dissolution with the proximal end of the first phalanx hollowed out to receive the rounded end of the metacarpal The proximal interphalangeal joint of the middle finger shows bony ankylosis of the type seen in rheumatoid arthritis

which had been decalcified by limited use, and the hemophilic hemorrhage progressed to cause an area of aseptic pressure necrosis in the bone

TRAUMATIC ARTHRITIS

A direct blow to a joint may produce effusion generally with some hemorrage, hemarthrosis was present in 93 per cent of 416 cases of severe injury

were treated, all again having complete subjective remissions Objective improvement in range of motion, was maintained in 13 of these patients Follow-up roentgenograms showed definite progression in over one-half of this group.

In Group II (24 patients) 11 had complete subjective remission and 7 additional patients received incomplete relief of symptoms. The range of motion of the spine was improved in 15 patients There was definite progression of roentgen involvement in 21 of these 24 patients

In Group III (13 patients) 11 received some degree of symptomatic relief; improvement in range of motion was reported in only 3 patients Definite progression of the roentgen findings was shown in all cases.

This carefully conducted study indicates that the remissions induced by this form of therapy appear to differ in no important respect from the spontaneous remissions of this disease and that it may be followed by asymptomatic progression of roentgen changes.

Smith, Boland and Hench[118] reported a comparative study of 75 patients with rheumatoid spondylitis treated with roentgen therapy and postural [illegible]

physical therapy, postural exercises, orthopedic measures and adequate diet.

Technique.—The technique of irradiation employed by various investigators has varied greatly [41, 97, 145, 152, 159] Some authors have stated that results depend a great deal on small changes in physical factors of irradiation This seems unreasonable to most students of this problem The method employed by most roentgenologists is a form of local high voltage roentgen therapy, as yet there is no recognized standard technique of irradiation for rheumatoid arthritis of the spine The treatment we have employed is similar to the technique of Kahlmeter,[68] Kemen,[71] Swaim,[134] and Hare,[47] although certain factors are different In recent years several groups have reported equally beneficial results and with fewer ill effects with smaller amounts of roentgen therapy

The technique used by Spishakoff[164] is as follows: In people of average [illegible]

target skin distance, and an output of approximately 45 r per minute, 200 r. (in air) are delivered to each field on alternate days, for a total of 600 r (air) per field After a rest period of four weeks, a second identical course of treatment is given, and after a second rest period of twelve weeks, the third and final course of treatment is administered

Scott reported excellent results with "wide-field" roentgen therapy in patients with rheumatoid spondylitis He used a special wide-angle x-ray tube to deliver a "roentgen-ray bath" to the whole trunk, front and back, which are covered by two over-lapping 12-inch fields The physical factors were. 140 kv, 3 mm aluminum filter and 60 to 100 r, with a standard distance of tube to the patients of 17 inches The first series of treatments consisted of 2 per week for 8 sessions; following a rest interval of six weeks, a second series included 2 treatments per week for 6 sessions and following

to knee joints reported by Mauck.[83] Hemorrhagic synovial fluid is roentgenographically homogenous; if free fat and blood are present the fluid is not of uniform density but tends to separate into layers.[91] In lateral

Traumatic arthritis may resemble degenerative joint disease radiologically.[5] To determine whether a progressive articular lesion has resulted from acute injury, repeated roentgenographic examinations are necessary.[6]



FIG. 35 —Multiple foreign bodies in the knee joint, most of which are located in the suprapatella bursa Note the hypertrophic spurs at the lower pole of the patella and along the joint margins *A*, Anterior posterior view *B*, Lateral view

Repeated long continued trauma to joints, such as the use of pneumatic tools or repeated damage sufficient to cause a scar to articular cartilage or other internal derangements may produce secondary traumatic osteoarthritis In individuals whose occupation requires long standing, the ... frequently are

... may development within several weeks at the site of injury or at a distance from it; this is shown in the roentgenogram as a characteristic patchy atrophy of bone.[25,132] This condition is not uncommon after acute trauma to a small joint (a wrist or an ankle). The bones of the adjacent foot or hand may become rarefied and marked vasomotor and trophic changes in the skin and muscles accompany the bone atrophy. Rarefaction of bone in such injured extremities involves both the cortex and the spongiosa.

another interval of six weeks, a third series of weekly treatments was giv
for six weeks. His method has been tried extensively in many clinics
England and there is little doubt that the results are not as good as wh

rapidly subside and frequently the calcium deposits disappear (Fig. 4
In chronic cases relief of pain may require a series of treatments

Chapman[24] in a review of 54 cases of bursitis of the shoulder treated wi
roentgen rays reports that when calcification was present, cure was effect
in 77 per cent. In those cases without calcium deposits the incidence

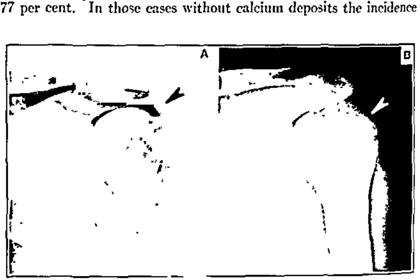


FIG 42 —*A*, Supraspinatus calcification located just above the junction of the great
tuberosity and head of humerus *B*, Following roentgen therapy the symptoms rapid
subsided and three months later the calcium deposits had disappeared

cure was only 58 per cent. The earlier the treatment was given the bett
the end results, relief in 90 per cent of cases followed when treatments we
started within thirty days of the onset of the symptoms. The onset
relief from pain in the acute cases occurs in the majority of cases with
seventy-two hours.

The average disability in 25 cases of bursitis treated with roentge
therapy by Klein and Klemes[74] was ten days. A similar group of 25 cas
treated by other means had an average disability of fifty days Pohle an
Morton[97] treated 69 cases of subdeltoid bursitis (50 per cent with calcifica
tion) with satisfactory relief of symptoms in 84 per cent of cases. McIntosh
treated 44 cases of painful shoulder due to rotator tendinitis, and reporte
relief of symptoms in 39 or 88 per cent.

The technique of roentgen therapy recommended by Dr. Eugene P
Pendergrass is as follows: *Acute or subacute*—Fifty r is given over th

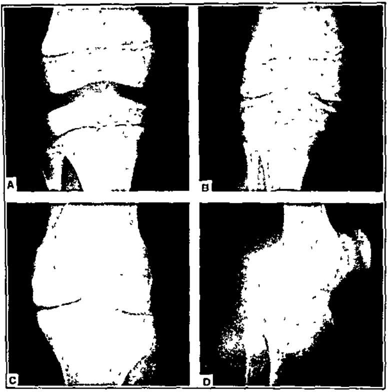


FIG 36 —Hemophilic arthritis Above Normal right knee (*A*) Involved left knee (*B*) in a boy nine years old with symptoms in the knee for several years Note the narrowing and irregularity of the joint space of the left knee The epiphysis on the involved side shows hypertrophy Below Adult knee in a patient age thirty-eight with chronic hemophilic arthritis for twenty years; anterior-posterior (*C*) and lateral views (*D*) Note the narrow joint space and sclerosis of the bones

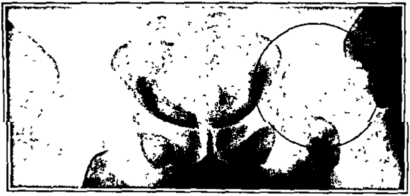


FIG 37 —Patient, aged thirty-four years, with swollen joints and limited motion. *Hemophiliac* Patient had severe discomfort in the right hip for twenty years Late stage of *hemophilic arthritis* of the hip

Traumatic Arthritis
1. [illegible]
liferation of bone.
4. In *chronic traumatic arthritis* due to recreation or posture, the early findings are usually negative except for a small swelling occasionally (as the tennis wrist); later, hypertrophic changes usually appear (as in prize-fighters).[22]

A diagnosis of Sudeck's atrophy cannot be made on the roentgenograms alone. The degree of osteoporosis does not parallel the patient's symptoms and frequently osteoporosis persists long after the syndrome has subsided.[10]

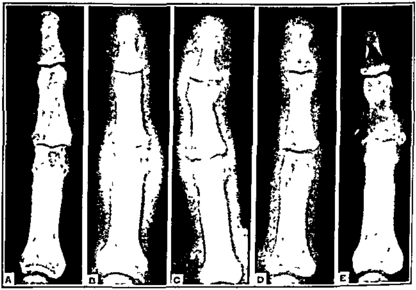


FIG. 38.—Punched out lesions in fingers. Localized cystic areas in bone occur in a variety of conditions: *A*. Rheumatoid arthritis; *B*. Gout; *C*. Osteoarthritis; *D*. Sarcoidosis, *E*. Metastatic carcinoma from bronchus.

GOUT

The characteristic feature of gouty arthritis is the deposition of monosodium urate crystals (which are transparent to x-rays) in the bone in the immediate neighborhood of the joints. These are seen on the film as punched-out areas on the surface of the articular bone, just beneath the cartilage or they may appear as holes in the articular end of the bone (Fig. 38*B*). They vary greatly in size from minute areas to ones large enough to resemble a bone cyst.

improvement occurred in about two-thirds of the patients and objective improvement in one-fourth Swaim concluded that the earlier roentgen therapy is begun in rheumatoid arthritis, the more rapid is the benefit, children seemed to improve more rapidly than adults. In his experience, frequent small doses over a long period of time resulted in more improvement than larger doses given infrequently or at longer intervals.

ev

gr

follows: for knee joints or small joints, 118 kilovolt peak, 5 milliamperes, 1 mm. aluminum filter, 14 inches distance, total of 225 r (given at the rate of 90 r per minute; for spines, 130 kilovolt peak, 5 milliamperes, 3 5 mm aluminum filter, 14 inches distance, total of 195 r (39 r per minute for five minutes), for degenerative joint disease of the knees and hips, 130 kilovolt peak, 5 milliamperes, 3 mm. aluminum filter, 14 inches distance, 107½ r (39 per minute for two and three-quarters minutes). They concluded that this form of therapy relieved pain in degenerative joint disease and has been of benefit to hips in rheumatoid spondylitis, and has been helpful locally in early cases of rheumatoid arthritis if given adequately

Osteo-Arthritis.—The relief of symptoms in patients with osteo-arthritis have, in general, been unsatisfactory; a few favorable reports have been

Kuhns and Morrison[156] reported on 118 patients with osteoarthritis; marked improvement occurred in 16 per cent, moderate improvement in 40 per cent and slight improvement in 29 per cent. These results are better than those obtained by most writers. Hernaman-Johnson[153] reported an analysis of 100 cases of osteoarthritis of the hips treated with x-rays and concluded that between one-quarter and one-third of these patients received striking benefit following the treatment.

Rationale.—The mechanism of action of roentgen therapy in arthritis is unknown. Following roentgen therapy, there occurs a marked dilatation of the capillaries, subcapillary plexus and arterioles under the area treated, which persists not only for a few hours (as occurs with other forms of heat), *but lasts for at least several days. This active hyperemia may be part of the answer to the improvement from roentgen therapy in these patients.*[26] Leedy believes that the beneficial effects of roentgen therapy in arthritis are due chiefly to destruction of leukocytes in the inflammatory process.[78]

Ill Effects.—The only potentially serious ill effect is leukopenia Transient decreases in the number of circulating leukocytes commonly occur and in an occasional case may decrease to 1,100 cells per cubic millimeter. These reactions are guarded against by weekly blood counts or more frequently if the count is low Symptoms referable to the gastro-intestinal tract frequently produce sickness. Anorexia, nausea and vomiting occur as a rule when the lumbar and dorsal part of the spine are treated. These symptoms usually begin four to six hours after therapy and always com-

and fewer ports

Roentgen Signs of Gout

[illegible]

articular surface or cystic areas may appear in articular bone Cartilage and bone are destroyed and small or large punched-out areas may appear [22]

5. In later stages, one may see irregular, asymmetrical *tophi* (in the soft tissue), bony hypertrophy and destruction of the articular ends of bones and cartilage with areas of erosion of varying size



Fig 39 —X-ray of great toe in a patient with gout Marked destructive changes are present in the metatarsophalangeal and interphalangeal joints typical of advanced gouty arthritis Blood uric acid was markedly elevated

29 Dawson, M. H : Chronic Arthritis, Nelson Loose Leaf Medicine, New York,

201, 232, 1941

36 Erdheim, J.: Über Wirbelsäulenveränderungen bei Akromegalie, Virchows arch f path Anat, 281, 197, 1931.

37. Ferguson, A B · Jour Bone and Joint Surg, 18, 297, 1936

38 Fineman, S

39 Frank, C H ·

40 Freiberg, J. A

41 Freyberg, R

42

43

44

45

Review Publishing Company, 1941

46 Haden, R L · Proc Interstate Postgrad Med Assoc North Am (1939), 29, 1940

47 Hare, H. F New England Jour Med, 223, 702, 1940

48 Harmon, P. H · Penna Med Jour, 45, 918, 1942

49 Harkness, A H Med Press, 207, 60, 1942

50 Hatch, P P. Brit Jour Radiol, 17, 70, 1944

51 Hefke, H W, and Turner, V D Jour Bone and Joint Surg, 24, 857, 1942

52 Hench, P S Ann Rheum Dis, 2, 19, 1940

53 ——· Jour Lab and Clin Med, 21, 524, 1936

54 Hench, P S, Vanzant, F R, and Nomland, R Trans Assn Am Phys, 43, 217, 1928

55 Hench, P S, et al Ann Int Med, 10, 754, 1936

56 —— Ann Int Med, 28, 66, 1948

Buckley,

66 —— Reports on Chronic Rheumatic Diseases, London, H K Lewis and Company, Ltd, 1939

67. Kaplan, I I New York State Jour Med, 45, 1339, 1945

68 Katz, S, Cake, C P, and Reed, H R New England Jour Med, 229, 498, 1943.

69 Keller, H : Med. Rec, 141, 76, 1935

70 Kelman, H.· Am Jour Surg, 64, 183, 1944

71 Kemen, A Verhandl d deutsch Gesellsch f inn Med Abstr, 49, 46, 1937

72 Key, J A Ann Surg, 95, 198, 1932

73 Key, J. A, and Large, A M Jour Bone and Joint Surg, 24, 281, 1942

74 Klein, I, and Klemes, I S Radiology, 37, 325, 1941

75 Kleinberg, S Jour Am Med Assn, 89, 172, 1927

76 Kuhns, J G · New England Jour Med, 219, 516, 1938

77 Langer, H. Radiology, 20, 78, 1933

78 Leddy, E T · Jour Am Med Assn, 115, 2210, 1940

79 Leventhal, D H. Surg Clin North Am, 23, 181, 1943

80 Lubert, M Am Jour Roentgenol, 51, 669, 1944

81 Ludwig, A O, Bennett, G A, and Bauer, W Ann Int Med, 11, 1248, 1938

Roentgen Signs of Gout (*Continued*)
6 Gouty changes are not often seen in larger joints; they do not occur in the shafts of the bones.
7. A number of joints of the hands or feet may show gouty changes while the remainder appear quite normal
8 Always *examine the great toe* if gout is suspected as it is affected sooner or later in about 80 per cent of cases
9 **Do not rule out gout if the x-ray is negative.**
10 Most suggestive roentgen findings *punched-out areas* 5 mm. or more in diameter in the subchondral bone of the base or head of the phalanges of the hands or feet



FIG 40.—*Synovial calcification in the suprapatellar bursa*, slight hypertrophic changes involving the knee joint in a patient aged sixty-six years

twenty-five years [64] In all suspected cases of gout, the feet should always be x-rayed for punched-out areas; these are usually 5 mm. or more in diameter and are found in the subchondral bone of the base of head of the phalanges of the hands or feet.[81] These are the most suggestive roentgen

diseases. Thorium x is said to be effective in psoriatic arthritis and in the late stages of rheumatoid spondylitis. It must be used with great care; complete blood counts should be made at frequent intervals. It has been injected intravenously at weekly intervals for a series of 8 increasing doses *not exceeding a total of 800 micrograms (if the products of disintegration*

with "chronic arthritis," using alpha-ray therapy, with a fatty carrier (neutral anhydrous lanolin) as a vehicle for the radon.[63] The local applications of radon ointment to 86 patients with a variety of articular and nonarticular rheumatic diseases were considered useful.[82] Kaplan[67] investigated this method of irradiation in a group of arthritis patients and concluded that, "under careful supervision and scientific procedures this form of therapeusis in our hands proved absolutely ineffective and useless for arthritic conditions."

Radio-active substances should not be employed by the general practioner Their use in patients with arthritis is still in the early experimental stage

BIBLIOGRAPHY

1 BAIRD, L W. Radiology, 37, 316, 1941
2 BAKER, L D Arch. Phys Med, 26, 389, 1945
3 BALENSWEIG, I. Surg Clin North Am, 23, 497, 1943
4 BAUER, E L Jour. Med Soc New Jersey, 38, 521, 1941
5 BAUER, W, BENNETT, G A, and ZELLER, J W. Trans Assoc Phys, 56, 349, 1941
6 BENNETT, G A Clinics, 1, 1448, 1943
7
8
9
10
11
12
13
14
15
16 and A. Churchill, 1934
17 BUCKLEY, C W. Ann Rheum Dis, 7, 17, 1948
18 BUCKLEY, E. J U S Nav Med. Bull, 44, 947, 1945
19 CACCIA, F. Arch ital chir, 18, 686, 1927
20 CAFFEY, J Pediatric X-ray Diag, Jr. Bk Publishers, Chicago, 1945.
21. CAMP, J D Radiology, 26, 339, 1936.
22 —— Ann Rheum Dis, 3, 145, 1943
23 CAMP, J D, and COVENTRY, M. B U S. Nav. Med Bull, 42, 56, 1943.
24 CHAPMAN, J F California and West Med, 56, 248, 1942
25 CHARCOT, J M 1868
26 COMROE, B I
27. COOPER, W
28 CRAIN, D C 1.

[illegible] New York,

[illegible]

35 Editorial. Jour Am Med Assn, 108, 1342, 1937
36 ERDHEIM, J: Über Wirbelsäulenveranderungen bei Akromegalie, Virchows arch f path Anat, 281, 197, 1931
37 FERGUSON, A B Jour Bone and Joint Surg, 18, 297, 1936
38 FINEMAN, S Med Clin North America, 21, 1641, 1937
39 FRANK, C H. Tri-State Med Jour, 15, 2925, 1943
40 FREIBERG, J A Jour Am Med Assn, 115, 2210, 1940
41 FREYBERG, R H Med Clin North Am., 30, 603, 1946
42 FRIED, C Strahlentherapie, 49, 634, 1934
43 GARLAND, L H Radiology, 25, 416, 1935
44 GELBER, L J, and GOLDBERG, S A: Radiology, 23, 45, 1934
45 GOLDHAMER, K X-ray Therapy of Chronic Arthritis, Quincy, Ill, Radiologic Review Publishing Company, 1941
46 HADEN, R L: Proc Interstate Postgrad Med Assoc North Am (1939), 28, 1940
47 HARE, H F New England Jour Med, 223, 702, 1940
48 HARMON, P H Penna Med Jour, 45, 948, 1942
49 HARKNESS, A H Med Press, 207, 60, 1942
50 HATCH, P P: Brit Jour Radiol, 17, 70, 1944
51 HEFKE, H W, and TURNER, V D Jour Bone and Joint Surg, 24, 857, 1942
52 HENCH, P S Ann Rheum Dis, 2, 19, 1940
53 ——— Jour Lab and Clin Med, 21, 524, 1936
54 HENCH, P S, VANZANT, F R, and NOMLAND, R Trans Assn Am Phys, 43, 217, 1928
55 HENCH, P S, et al Ann Int Med, 10, 754, 1936
56 ——— Ann Int Med, 28, 66, 1948
57 HENSON, E B West Virginia Med Jour, 32, 83, 1936
58 HERTZBERG, J. Acta Radiol, 26, 484, 1945
59 HORWITZ, T: Arch Int Med 65, 1178 1940
60 HOWORTH, M B Surg, Gynec and Obst, 80, 337, 1945
61 JANSEN, K F Acta Radiol, 24, 285, 1943
62 JONES, O O: New Orleans Med and Surg Jour., 93, 363, 1941
63 [illegible]
64 [illegible] 3, 1944
65 [illegible] C W Buckley, [illegible]
66 [illegible] H K Lewis and Company, [illegible] 1939
[illegible] Med, 45, 1339, 1945
[illegible] R New England Jour Med, 229, 498, [illegible]
69 KELLER, H Med Rec, 141, 76, 1935
70 KELMAN, H Am Jour Surg, 64, 183, 1944
71 KEMEN, A Verhandl d. deutsch Gesellsch f inn Med Abstr, 49, 46, 1937
72 KEY, J A: Ann Surg, 95, 198, 1932
73 KEY, J A, and LARGE, A M Jour Bone and Joint Surg, 24, 281, 1942
74 KLEIN, I, and KLEMES, I S Radiology, 37, 325, 1941
75 KLEINBERG, S Jour Am Med Assn, 89, 172, 1927
76 KUHNS, J G: New England Jour Med, 219, 516, 1938
77. LANGER, H: Radiology, 20, 78, 1933
78 LEDDY, E T Jour Am Med Assn, 115, 2210, 1940
79 LEVINTHAL, D H Surg Clin North Am, 23, 181, 1943
80 LUBERT, M Am Jour Roentgenol, 51, 669, 1944
81 LUDWIG, A O, BENNETT, G A, and BAUER, W Ann Int Med, 11, 1248, 1938

PART II

RHEUMATOID ARTHRITIS

CHAPTER 8

THE ETIOLOGY OF RHEUMATOID ARTHRITIS

By CHARLES RAGAN, M D

THE *cause of rheumatoid arthritis remains unknown* There are certain factors upon which general agreement is present [9,35] (1) Rheumatoid arthritis is a disease of temperate climates with a low incidence in the tropics [5] (2) It may have a tendency to be familial, which, however, is difficult to trace genetically (3) Eighty per cent of the cases occur between the ages of twenty and fifty with the peak at thirty-five to forty (4) Females are more commonly affected than males in a ratio of 2 or 3 1 (5) Investigation shows that, in the period preceding the onset of symptoms, emotional shocks are common and this is often manifested by a severe depression [52] (6) Pregnancy,[11,23] or (7) Involvement of the liver as in infectious hepatitis with or without jaundice leads to a more or less temporary remission in a high percentage of patients [21,24]

The Initial Lesion in Rheumatoid Arthritis—a Clue to Etiology?—Since 1929,[32] indirect evidence has accumulated that in both rheumatic fever and rheumatoid arthritis the initial lesion may involve primarily the interfibrillar substance of the connective tissue [36] The nature of this material is not well understood, although it is presumably a mucopolysaccharide in combination with a protein. In the hypothesis advanced by this theory, changes in the cellular components are presumed to be secondary to a change in the interfibrillar material but the nature of this is not understood. The composition of two of the mucopolysaccharides found in interfibrillar material is known: (1) chondroitin sulfuric acid, and (2)

subchondral plate and on the surface of the joint. Alterations in the hyaluronic acid of joint fluid have been shown to be present in active rheumatoid arthritis,[19,45] consisting of depolymerization of the hyaluronic acid and an increase in total amount of hyaluronic acid present,[40] which points to a defect in synthesis of this mucopolysaccharide The inflam-

infiltration, fibrosis and scarring The theory remains to be proved, and

diseases. Thorium x is said to be effective in psoriatic arthritis and in the late stages of rheumatoid spondylitis. It must be used with great care;

lea

with "chronic arthritis," using alpha-ray therapy, with a fatty carrier (neutral anhydrous lanolin) as a vehicle for the radon.[63] The local applications of radon ointment to 86 patients with a variety of articular and non-articular rheumatic diseases were considered useful.[62] Kaplan[67] investigated this method of irradiation in a group of arthritis patients and concluded that, "under careful supervision and scientific procedures this form of therapeusis in our hands proved absolutely ineffective and useless for arthritic conditions."

Radio-active substances should not be employed by the general practioner Their use in patients with arthritis is still in the early experimental stage.

I am grateful to J F Holt, M D, of the Department of Roentgenology, and W D. Robinson, M D, Rackham Arthritis Research Unit, University of Michigan, Ann Arbor, Michigan, for roentgenograms of cases illustrating Boeck's sarcoid, Hemophilia, Gout and Psoriatic arthritis

BIBLIOGRAPHY

1 BAIRD, L W Radiology, 37, 316, 1941
2 BAKER, L D Arch Phys Med, 26, 389, 1945
3 BALENSWEIG, I Surg Clin North Am, 23, 497, 1943
4 BAUER, E L Jour Med Soc. New Jersey, 38, 521, 1941
5 BAUER, W, BENNETT, G A, and ZELLER, J. W.. Trans Assoc Phys, 56, 349, 1941
6 BENNETT, G A Clinics, 1, 1148, 1943
7 ...2
8
9
10 ...44
11 BOLAND, E W, and PRESENT, A J: Jour Am Med. Assn, 129, 843, 1945
12 BORAK, J Radiology, 47, 128, 1946
13 BORAK, J, and Taylor, H K.: Radiology, 45, 377, 1945
14 BOSWORTH, B M Jour Am Med. Assn, 116, 2477, 1941
15 BOYD, D Jour Bone and Joint Surg, 16, 671, 1934
16 BRAILSFORD, J F Radiology of Bones and Joints, London, J and A Churchill, 1934
17 BUCKLEY, C W.. Ann Rheum Dis, 7, 17, 1948
18 BUCKLEY, E J · U S Nav Med Bull, 44, 947, 1945.
19 CACCIA, F. Arch ital chir, 18, 686, 1927
20 CAFFEY, J Pediatric X-ray Diag, Jr Bk. Publishers, Chicago, 1945
21 CAMP, J D. Radiology, 26, 339, 1936
22 ——— Ann Rheum Dis, 3, 145, 1943
23. CAMP, J. D, and COVENTRY, M B · U S Nav Med Bull, 42, 56, 1943
24 CHAPMAN, J F California and West Med, 56, 248, 1942
25 CHARCOT, J. M Arch de physiol norm. et path, 1, 161, 1868
26 COMROE, B I Jour. Am Med. Assn, 117, 830, 1941
27. COOPER, W · Jour Bone and Joint Surg, 24, 114, 1942
28 CRAIN, D C Med Ann District of Columbia, 10, 293, 1941.

is of little assistance in uncovering the etiology of the disease since the cause of these alterations is completely unknown.

Rheumatoid Arthritis—an Infectious Disease?—Rheumatoid arthritis has the characteristics of an inflammatory process—fever, tachycardia, rapid sedimentation rate, leukocytosis, lymphadenopathy, occasionally splenomegaly and inflamed articular structures. Competent bacteriological investigations of blood, synovial fluid, synovial tissue and subcutaneous nodules[1] have shown no causative infectious agent. It has not been possible to transmit the disease by intraarticular injection of joint fluid from patients with rheumatoid arthritis to other human subjects.[34] Homologous subcutaneous nodules have failed to survive on transplantation.[2] The inflammatory features suggesting an infectious origin are seen in acute gout, a metabolic disorder, and in rheumatic fever, probably a reaction to sensitization. The association of rheumatic fever with Group A hemolytic streptococci seems to be valid and is manifested in a very high percentage of cases by a preceding upper respiratory infection and an elevated antistreptolysin titer. In rheumatoid arthritis a preceding history of upper respiratory infection, and an elevated antistreptolysin titer occur in about 20 per cent of the patients.[49] This is considered to be a co-incidental finding and of no etiologic significance. Certain serologic reactions with Group A hemolytic streptococci are found in rheumatoid arthritis. The sera of from 50 to 75 per cent of patients with the disease will agglutinate Group A hemolytic streptococci in a particular fashion,[8,37] whereas under carefully controlled conditions, the agglutination is rarely seen in patients other than those with rheumatoid arthritis. Serologic reactions in the sera of patients with rheumatoid arthritis to various carbohydrate and protein components of the hemolytic streptococcus parallel the agglutination of the whole organism but to a lesser degree,[29] particularly when collodion particle technique is used.[4,55] Sheep cells which have been sensitized with one-half the agglutinating titer of anti-sheep cell rabbit serum are also agglutinated by the sera of patients with rheumatoid arthritis and a test has been devised using the differential titer between sensitized and normal sheep cells.[46] The differential titer is measured as the reciprocal of the dilution and a titer of 16 or more is seen infrequently except in rheumatoid arthritis. This test is more easily performed than, but mirrors, the agglutination of Group A hemolytic streptococci. It had

serum than with normal serum because of a substance present in the gamma-beta globulin fraction of the former. These tests are of some diagnostic help but so far have given no inkling of the etiology.[4]

The pendulum has now swung rather completely away from the previously accepted thesis that rheumatoid arthritis is associated with focal infection. Many patients do not have obvious foci of infection. If such a focus is found, the removal of it has not influenced the course of the disease. A focus of infection, if severe, acts as a drain upon the patient's general health, but is of no significance in the causation or the maintenance of activity of the disease.

29 DAWSON, M H. Chronic Arthritis, Nelson Loose Leaf Medicine, New York, Thomas Nelson and Sons, 5, 605, 1935
30 DE TAKATS, G: Arch Neurol and Psychiat, 1, 318, 1943
31 DOUB, H. P.: Radiology, 24, 391, 1935
32 ——: Am Jour Roentgenol, 30, 26, 1933
33 DUNHAM, C L., and KAUTZ, F G. Am Jour Med Sci, 201, 232, 1941.
34 DUSCHAK, E T.: Arch Phys Therap, 23, 95, 1942
35 Editorial: Jour. Am Med Assn, 108, 1342, 1937
36 ERDHEIM, J. Uber Wirbelsaulenveranderungen bei Akromegalie, Virchows arch f path Anat, 281, 197, 1931
37 FERGUSON, A B: Jour Bone and Joint Surg, 18, 297, 1936.
38. FINEMAN, S: Med Clin North America, 21, 1641, 1937.
39 FRANK, C H Tri-State Med Jour, 15, 2925, 1943
40 FREIBERG, J A Jour. Am Med Assn, 115, 2210, 1940
41 [illegible]

1940
47 HARE, H I. New England Jour Med, 223, 702, 1940
48 HARMON, P. H Penna Med Jour, 45, 918, 1942
49 HARKNESS, A H Med Press, 207, 60, 1942
50 HATCH, P. P Brit Jour Radiol, 17, 70, 1944
51 HEFKE, H W, and TURNER, V D Jour Bone and Joint Surg, 24, 857, 1942
52 HENCH, P S: Ann Rheum Dis, 2, 19, 1940
53 ——: Jour Lab and Clin Med, 21, 524, 1936
54 HENCH, P S, VANZANT, F R, and NOMLAND, R Trans Assn. Am Phys, 43, 217, 1928
55 HENCH, P S, *et al* Ann Int Med, 10, 754, 1936
56 —— Ann Int Med, 28, 66, 1948
57 HENSON, E B [illegible] 32, 83, 1936
58 HERTZBERG, J [illegible]
59 HORWITZ, T A[illegible]
60 HOWORTH, M B [illegible]
61 JANSEN, K F Acta Radiol, 24, [illegible]
62 JONES, O O New Orleans Med and Surg Jour., 93, 363, 1941.
63 J[illegible]
64 J[illegible] 3, 1941
65 K[illegible] C W [illegible]
66 — [illegible] H K [illegible]
Company, Ltd, 1939
67. KAPLAN, I I. New York State Jour Med, 45, 1339, 1945
68 KATZ, S, CAKE, C P, and REED, H R New England Jour [illegible]
1943
69 KELLER, H Med Rec, 141, 76, 1935
70 KELMAN, H. Am Jour Surg, 64, 183, 1944
71 KEMEN, A Verhandl d deutsch Gesellsch f inn Med [illegible]
72 KEY, J. A. Ann Surg, 95, 198, 1932
73 [illegible] Jour Bone and Joint Surg, [illegible]
[illegible] gy, 37, 325, 1941.
[illegible] 89, 172, 1927
[illegible] 1, 219, 516, 1938
77 LANGER, H Radiology, 20, 78, 1933
78 LEDDY, E T Jour Am Med Assn, 115, 2210, 1940
79 LEVINTHAL, D H Surg Clin North Am, 23, 181, 1943
80 LUBERT, M Am Jour Roentgenol, 51, 669, 1944
81 LUDWIG, A O, BENNETT, G A, and BAUER, W: Ann [illegible]
1938

82 Lustig, F. Med Rec, 158, 225, 1945.
83. Mauck, H. P.. Am Jour. Surg, 56, 54, 1942.
84. McDonald, E. J, and Lozner, E. L.. Am. Jour. Roentgenol, 49, 405, 1943
85 McEwen, C., and Thomas, E W.: Med. Clin. North Am, 22, 1275, 1938
86 McLaughlin, H L.. Jour. Bone and Joint Surg, 26, 31, 1944.
87. Mitchell, J. K.· Am. Jour. Med. Sci, 8, 55, 1931.
88 Moritz, A. R.: Virchows Arch f path Anat, 267, 746, 1928.
89. [illegible]
90. [illegible]
91. [illegible]
92 [illegible]
93. [illegible]
94 [illegible]
95 [illegible]
96. [illegible] 118, 859, 1942
97. Pohle, E. A, and Morton, J. A · Radiology, 49, 19, 1947.
98 Pomeranz, M M: Am. Jour. Roentgenol and Rad Ther, 29, 753, 1933
99. Pomeranz, M. M, Friedman, L. J, and Tunick, I. S.: Radiology, 37, 295, 1941
100 Porter, W. B, and Rucker, J E. Jour. Am. Med Assn, 92, 1513, 1929
101. Quaintance, P A: Jour Bone and Joint Surg, 20, 353, 1938
102. Raymond, F., Janet, J, and Levy, H.. Bull et mem. Soc med. d hôp de Paris, 47, 200, 1923.
103 Rechtman, A M: Surg, Gynec and Obst, 49, 683, 1929
104. [illegible]
105 [illegible] 33
106 [illegible]
107 [illegible] Med, 69, 238, 1942.
108 Rost, E R: Brit Med Jour, 2, 978, 1921
[illegible] 936.
[illegible] Newnes, Ltd, 1939
113 ———. Radiological Atlas of Chronic Rheumatic Arthritis, London, Oxford University Press, 1935
114. ———· Rheumatism, 1, 1, 1938.
115 Shapiro, M J.· Jour Am. Med Assn, 98, 1965, 1932.
116 Short, C L.: Med Bull North African Theat. Op, 1, 10, 1944.
117. Short, C. L, and Bauer, W.: New England Jour. Med, 225, 145, 1941
118. Smith, R. T, Boland, E. W., and Hench, P. S: Ann. Rheum Dis, 6, 114, 1947.
119 Smyth, C J, Freyberg, R H, and Lampe, I. Jour Am Med Assn, 117, 826, 1941.
120. Smyth, C J, Freyberg, R H, and Peck, W. S. Jour Am. Med. Assn, 115, 2209, 1940
121 ———· Jour Am. Med Assn, 116, 1995, 1941.
122 Sokolo [illegible]
123 Spackm [illegible] 936.
124 [illegible]
125 Spink, [illegible] 1945
126. Staunic [illegible]
127. Steche [illegible]
128. Stein, I: Am Jour Roentgenol, 37, 678, 1937.
129. Steindler, A: Urology and Cutaneous Rev., 49, 568, 1945
130 Steven, G. O: Ann Rheum Dis, 6, 184, 1947.
131 Stuart, B M.· Am Jour. Med Sci, 208, 717, 1944
132 Sudeck, P.· Deutsch. med Wchnschr, 28, 170, 1902
133 Sutherland, C. G Radiology, 4, 355, 1925.
134. Swaim, L. T.· Jour Am. Med Assn., 115, 2209, 1940.

is probably the result of the union of antigen and antibody on the cell surface while in the latter the lesion results from endothelial cell damage with increased vascular permeability, vascular occlusion and infarction. Entirely similar arterial lesions have been seen in hypersensitivity of the serum sickness type, in periarteritis nodosa, and in patients who have died shortly after a hypersensitive reaction to a sulfonamide.[42] In most patients with periarteritis nodosa, however, no such antigen has been detected

There has been a great deal of indirect evidence—animal and clinical—to show that rheumatic fever may be considered to be an allergic reaction to products liberated in a sensitized individual. A previous infection with Group A hemolytic streptococci[6] had been implicated as a possible sensitizing factor. It has been recently shown[41] that sensitization probably follows repeated infections with different types of Group A hemolytic streptococci, since type-specific immunity persists for long periods of time

The clinical similarities[13] between rheumatoid arthritis, rheumatic fever, lupus erythematosus [illegible]
to group these diseas [illegible]
or diffuse diseases of [illegible]
common etiology and in fact most investigators now feel that this is not the case In any event, although the lesions seen in rheumatoid arthritis are of an inflammatory nature and could well be the result of sensitization, no comparable joint lesions—particularly the self-perpetuating character of the pattern of inflammation—have been produced in experimental animals and no responsible antigen has been detected after vigorous search over a period of thirty years. The failure to recover an infectious agent and the failure of such a supposed agent to become manifest during long-continued administration of ACTH or cortisone in patients with rheumatoid arthritis have, however, again served to bring into focus the role of allergy in this disease. The antiphlogistic action of these hormones against the inflammatory process of rheumatoid arthritis, of serum sickness, and of lesions presumably of a hypersensitive character such as rheumatic fever and periarteritis nodosa is essentially similar. The anti-inflammatory properties of the hormones are, however, apparently completely non-specific in regard to the cause of inflammation since the inflammatory response is decreased to chemical, viral, bacterial and traumatic stimuli as well as to hypersensitivity

The similarity between the collagen degeneration seen in serum sickness and that seen in the rheumatoid nodule has been noted in favor of an allergic basis for the latter disease. By electron microscopy,[20,30] however, the collagen in a rheumatoid nodule is not altered and the amino acid hydroxyproline—a component of collagen—is not found in excess in fibrinoid tissue.[27] The present trend of thought indicates that the change probably takes place in the interfibrillar material lying between the collagen bundles and between the collagen fibrils as well. With changes in this material, collagen bundles swell, but the fibrils remain intact.

Rheumatoid Arthritis—a Psychosomatic Disease?—Discussion is now widespread concerning the role of emotional trauma in the initiation and maintenance of this disease One must consider both the emotional changes secondary to the disease and those existing prior to its onset [35] No adequate documentation of emotional factors alone in the causation of this disease has been produced but apparently remissions and exacerbations may

135 ———: Jour. Indiana Med Assn, 38, 487, 1945.
136 Taylor, G D, Ferguson, A. B, Kasabach, H, and Dawson, M H · Arch. Int Med, 57, 979, 1936
137. Widmann, B. P, and Stecher, W R · Radiology, 27, 541, 1936.
138 Wile, V. J, and Butler, M G · Jour Am Med Assn, 94, 1053, 1930
139 Williams, D I G.: Ann Rheum Dis, 7, 21, 1948
140 Wolin, I: Radiology, 42, 79, 1944
141. Woolf, D L.: Ann. Rheum Dis, 6, 208, 1947
142 Zulick, J D · Penna Med Jour, 44, 1284, 1941
143 Abel, M S. Radiology, 55, 235, 1950
144 Abramson, D J, and Kambero, S. Jour Bone and Joint Surg, 31A, 275, 1949

150 Gelber, L J: Int Rec Med, 164, 62, 1951
151 Guest, C M and Jacobson, H G · Am Jour Roent, 65, 760, 1951.
152 Hemphill, J E and Reeves, R J Am Jour Roent and Rad Ther, 54, 282, 1945
153 Hernaman-Johnson, F Rheumatism, 5, 44, 1949
154 Holt, J F. and Owens, W I Radiology, 53, 11, 1949.
155 Isaacson, A S and Whitehouse, W M Jour. Bone and Joint Surg, 31A, 306, 1949
156 Kuhns, J G and Morrison, S L New England Jour Med, 235, 399, 1946
157. Kuzell, W C Stanford Med Bull, 6, 324, 1948
158. McIntosh, H C Jour Am Med Woman's Assn, 3, 277, 1948
159 McWhirter, R Brit Jour Rad, 18, 302, 1945

166 Stursberg, H Deutsche med Wchnschr, 61, 5, 1935
167 Wade, P: Univ Leeds Med Mag, 17, 89, 1947.
168 Williams, A. J California Med, 70, 257, 1949

frequently be related to emotional stresses and strains.[25] The makeup of these patients is frequently that of an anxiety state. An understanding attitude on the part of the physician by which care is directed to the patient as a whole rather than to isolated segments of his anatomy or of

to subsequent tissue change. These patients need complete care and understanding of all their problems, physical, emotional, economic and social as is required in the proper management of every disease state.

Rheumatoid Arthritis—a Nutritional Disease?—The striking inanition seen in patients with far advanced rheumatoid arthritis has led many to presuppose that some factor concerned with food ingestion was lacking. Studies of the diet of these patients in the prodromal period have shown no obvious deficiencies.[8] No food allergy has been associated with rheumatoid arthritis.[28] No definite vitamin deficiency has been found although fairly uniformly a lowered plasma vitamin C level exists.[19] However, when this returns to normal after the administration of large amounts of vitamin C, no change in the course of the disease follows.[19] It has been shown that the use of vitamin D preparations has not materially affected the course of the disease.[19] Changes in serum proteins are chiefly in the nature of increased globulin which is now considered to be a manifestation of immunity such as is seen in other instances of chronic inflammation. Changes in carbohydrate tolerance[14] may be regarded as a result of the disease rather than as causative mechanisms. Changes in sulphur metabolism are variable and, if corrected by the injection of colloidal sulphur, the disease process continues unabated.[15] No alteration in calcium or phosphorus metabolism has been found in patients with rheumatoid arthritis.[4]

Rheumatoid Arthritis—a Nervous System Disease?—Various theories of imbalance of the central nervous system or of the sympathetic and parasympathetic systems have been advanced. The finding of perineural nodules[15] and lymphorrhages in the sarcolemma sheaths of skeletal muscle[16] has emphasized the question of dysfunction of the nervous system in this disease. To be noted as well is the observation that changes in a peripheral joint similar to that seen in rheumatoid arthritis may follow section of a peripheral nerve. Abnormal reactions in this sphere probably result from the disease rather than serve as causative mechanisms, since no definite causal relationship has been found by the usual clinical and laboratory methods between the onset of rheumatoid arthritis and diseases of the nervous system.

SUMMARY

In any given disease, one must consider two components: (1) the host, and (2) the noxious agent. In rheumatoid arthritis, precipitating factors appear to be varied and an hypothesis compatible with all the clinical data must presuppose, in part at least, that the host is at fault and that many different types of noxious stimuli may initiate the process now commonly called activity of the rheumatoid process.

CHAPTER 7

ORGANIZATION OF AN ARTHRITIS CLINIC

By Wallace Graham, M.D.

It is becoming more and more the conviction of rheumatologists that patients with arthritis respond best to treatment in a fully integrated rheumatism center, with hospital and out-patient facilities.[1]

The need for special arthritis centers for certain cases requiring prolonged therapy, comparable to the sanatorium in the treatment of tuberculosis, has already been recognized in many regions, notably in the Scandinavian countries and Great Britain.

The experience in rheumatism centers established during World War II has adequately shown that progressive cases respond unusually well to group therapy in a clinic where all facilities are available. Comparable results are difficult to obtain when such cases are scattered widely throughout the medical wards of general hospitals

The chief aims of an army rheumatism center were outlined by Hench and F

1. ere difficult
cases *ms with a*
special knowledge of rheumatic diseases.

2. Intensive treatment: To provide special facilities for the treatment of the more severe or progressive cases

3 Prompt disposition: To accomplish as great a reduction in hospitalization-time as is consistent with adequate treatment.

4. Increased salvage. To restore to work, if possible, more men with "cured" or "arrested disease."

5 Rehabilitation: To educate and rehabilitate those whose disability necessitates a change in occupation.

reduce
t tion in

The value of an arthritis center does not cease with the satisfactory progress of those undergoing treatment A unique opportunity is afforded for research and the study of hundreds of patients with rheumatic disease which should be a constant source of knowledge to the whole medical profession As well, an excellent opportunity for concentrated study is available to the interne or general practitioner who is seeking post-graduate instruction in the rheumatic diseases.

The staff of the ideal arthritis clinic should include the following:

A chief of staff: preferably an internist, with a good working knowledge of the diagnostic, therapeutic and research problems in this field (*i. e*, a *rheumatologist*).

REFERENCES

1 ANGEVINE, D M, ROTHBARD, S., and CECIL, R. L · Jour Am Med Assn, 115, 2112, 1940
2 BAUER, W., CLARK, W. S, and DIENES, L.: The Practitioner, 166, 5, 1951.
3 BAYLES, T. B, RICHARDSON, H, and HALL, F. C. New England Jour Med, 229, 319, 1943.
4. BOOTS, R. H, LIPMAN, M O., COSS, J A., JR., and RAGAN, C.· Rheumatic Diseases Based on the Proceedings of the 7th International Congress on Rheumatic Diseases, p. 336, Philadelphia, Pa, W. B Saunders, 1952.
5 CLARKE, J. T. Lancet, 1, 1169, 1915
6. COBURN, A. F. The Factor of Infection in the Rheumatic State. Baltimore, Md, Williams and Wilkins Co, 1931
7. COLLINS, D H, and GOLDIE, W: Jour. Path and Bact, 50, 323, 1940
8 DAWSON, M H, OLMSTEAD, M and BOOTS, R H: Jour. Immunol, 23, 187, 1932
9 DAWSON, M H, and RAGAN, C · Chronic Arthritis, Nelson's Loose Leaf Medicine, New York, Thomas Nelson & Sons, In Press
10 DOBRINER, K Proc. Amer. Rheum Assn, Ann Rheum Dis, 10, 464, 1951
11 EDSTROM, G. Acta Med Scand, 103, 90, 1940.
12 FINDLAY, G M, MACKENZIE, R. D, and MACCALLUM, F. O.: Brit Jour Exper Path, 21, 13, 1940
13 FLYNN, J., *et al* Amer Jour. Med. 1, 675, 1946
14 FLYNN, J E, and IRISH, O J. Science, 104, 344, 1946
15 FREUND, H A, STEINER, G, LEICHTENTRITT, B, and PRICE, A. E. Amer Jour. Path 18, 865, 1942.
16 FREUND, H A, *et al* Science, 101, 202, 1945
17. FREYBERG, R. H. Jour Am. Med Assn, 115, 2114, 1940
18 ——— Jour Am Med. Assn, 119, 1165, 1942
19 FREYBERG, R H, BLOCK, W. D. and FROMER, M F · Jour. Clin. Invest, 19, 423, 1940
20 GALE, J C: Amer Jour. Path, 26, 707, 1950.
21 GARDNER, F, STEWART, A, and MACCALLUM, F. O. Brit Med Jour, p. 677, Nov 1945
22. GLASER, R. J, *et al* Jour. Clin Invest, 30, 640, 1951
23 HENCH, P. S.· Proc Staff Meet, Mayo Clin, 13, 161, 1938
24 ———: Med. Clinics N A, 24, 1209, 1940.
25. JOHNSON, A, SHAPIRO, L B., and ALEXANDER, F: Psychosomatic Med, 9, 295, 1947
26. JORDAN, E P., *et al* Jour Am Med Assn, 119, 1089, 1942
27. KANTOR, T., *et al.*: Proc Amer. Rheum Assn, Ann. Rheum Dis, 10, 471, 1951.
28. KASS, E H, *et al.*· 2nd Clin ACTH Conf., 2, p 376, Blakiston Co, Philadelphia, Pa, 1951
29 KEEFER, C. S: New England Jour Med, 213, 644, 1935
30. KELLGREN, J. H., *et al*. Nature, 168, 493, 1951
31. KING, E Q, *et al.*: Jour. Amer. Med Assn., 147, 238, 1951.
32. KLINGE, F. and GRZIMEK, N: Virchow's Arch. f. path anat, 284, 646, 1932
33. KUZELL, W C, and MANKLE, E. A.. Proc. Soc Exper. Biol. & Med, 74, 677, 1950.
34 LEVINSKY, W. J, and LANSBURY, J Proc Soc Exper Biol & Med, 78, 325, 1951.
35 LUDWIG, A O: Bull. Rheum Dis., 2, #8, April 1952.
36 MEYER, K. Phys Rev 27, 335, 1947.
[illegible] Jour Clin Invest 10, 323, 1931
[illegible] ie American Rheuma-
[illegible]) and 1268, 1949
[illegible])19.
[illegible] t. Med, 76, 131, 1945

Physical Medicine)

An occupational therapist, to supervise retraining

Nurses, as necessary depending on the size of the clinic. They should be trained in the care of arthritic patients, and must appreciate the sympathy and encouragement which are necessary to maintain the morale of the arthritic patient.

One or more social workers: the importance of these aides will be discussed in detail below. A laboratory technician One or more research fellows

A Rehabilitation Counsellor: the need for such an agent is described below

A layman's committee, composed of citizens (especially women) who will be able to aid the clinic by raising funds, disseminating knowledge concerning the prevention and care of arthritis, etc

If it is possible, the arthritis clinic should have sufficient room available so that all of the consultants may be present at the same time in separate rooms to complete any necessary consultations at once; this obviates the necessity of asking the patient to return to the hospital on four or five different days for such consultations. If one individual in each specialty is assigned to the arthritis clinic, both the patient and the specialist will profit thereby. The patient will derive the benefit of seeing the same specialist for any revisits which may be necessary, rather than, for example, to see a variety of otolaryngologists on subsequent visits, some of whom may not be particularly interested in arthritis. The specialist will benefit by noting the [illegible]

are a *sine qua non* in the treatment of arthritis If funds are available, a therapeutic pool for underwater exercises should be included in the armamentarium of this department.

Occupational therapy is of great value in re-educating wasted muscles and in keeping the mind and limbs of the arthritic patient occupied.

It is especially advisable that sufficient funds be set aside, if possible, for the use of one or more *research fellows* in arthritis

In the *orthopedic* room, apparatus must be on hand for making plaster splints, body casts and other orthopedic appliances

A psychiatrist may play an important part in the adequate study of patients suffering from various forms of rheumatism Pain, aching and stiffness are frequently the result of emotional rather than organic processes. The basic problem may be deep seated and may require thorough psychiatric investigation (see Psychogenic Rheumatism).

The *social service worker* can take care of problems such as securing adequate relief funds for the patient (if necessary), obtaining orthopedic

44 Ropes, M W, Rossmeisl, E C, and Bauer, W Jour Clin Invest, 22, 785, 1943
45 Ropes, M W Ann. Rheum Dis, 7, 35, 1948
46. Rose, H, *et al* · Proc Soc. Exper Biol & Med, 68, 1, 1948
47. Sabin, A B: Science, 89, 228, 1939
48 Sclater, J G: Ann Rheum Dis, 3, 195, 1943
49 Selye, H, *et al*. Jour Am Med Assn, 134, 201, 1944
50 Schwartzman, G: Proc Soc Exper. Biol & Med, 79, 573, 1952
51 Southam, C. M, and Babcock, V I *Proc Soc Exper Biol & Med*, 78, 105, 1951.
52 Thomas, G. W Am. Jour. Psychiat, 93, 693, 1936
53 Thomas, L, and Good, R A: Proc Soc Exper Biol & Med, 76, 601, 1951
54 Venning, E H, Johnson, L G, and Rose, B 2nd Clin ACTH Conf, 2, p 49, Blakiston Co, Philadelphia, Pa., 1951
55 Wallis, A. D Amer Jour Med Sci, 213, 91, 1947

shoes, assisting in procuring attendants for children when the mother is confined to a hospital, etc. Many of the details which cannot be performed by the physician because of lack of time, can be accomplished by these trained workers.

The Rehabilitation Counsellor, often a trained social service worker, is essential for proper clinic care.

The great majority of patients who suffer even from the severe forms of arthritis may, after prolonged treatment, become useful and independent members of society, and only rarely should this affliction reduce them to dependency and helplessness. Since World War II a much more comprehensive and integrated concept of rehabilitation has been evolved in an effort to develop and restore the handicapped person to the fullest physical, mental, social, vocational and economic usefulness of which he is capable. This must be the primary aim of any arthritis center. It must be realized that a physical disability may not be an occupational handicap and that a disability such as arthritis is only *one* of a wide variety of factors which differentiate the working potential of one man from another. There can be no rehabilitation without employment so that industry must play a prominent part in the restoration of disabled people. A rehabilitation counsellor, familiar with placement problems is therefore essential to attain the aims of a Rheumatism Center An effort must be made to find employment which will not, through mental or physical stress, lead to aggravation and progression of the disease. It must be stressed that the old system of listing jobs that are suitable for a person with a specific disability, such as arthritis is now obsolete No two persons *with like* disability have like capability, and, of greater importance, an individual should not be hired because of his *disability* but because he has the required *ability* based on his capacity, his training, his experience, his aptitude and determination The patient must be considered as a 'whole' and not for example, as a 'man with arthritis in both knees'. The interest of the clinic must not cease when inflammation has subsided but only when the patient has been restored to the fullest usefulness of which he is capable.

The clinic must have available to it a number of *hospital beds* for special care when this is needed, as in acute arthritis, correction of deformities, operative procedures, etc

Every arthritis clinic should have an auxiliary committee of volunteers, whose function is to spread information concerning the working of the clinic, the prevention and incidence of arthritis, and to obtain additional funds for the assistance of the clinic and research in arthritis. A member of this committee should be present during the working hours of the clinic to observe its function and to assist the social service worker in any problems which may arise. Where additional medication or orthopedic appliances cannot be afforded in particular cases, it is the function of this committee to devise ways and means of obtaining these needed articles for the patients.

The clinic should have available reading matter on Rheumatic Diseases and allied subjects This material should include textbooks on the subject, the Arthritis Clinic Manual,[1] the "Primer on Arthritis,[3]" and some of the journals listed below.

CHAPTER 9

THE PATHOLOGY OF RHEUMATOID ARTHRITIS

By Edward F. Rosenberg, M.D.

INTRODUCTION

The pathologic lesions of rheumatoid arthritis are found widespread throughout the body. Joints are the main sites of destructive changes but important alterations occur in other organs as well. Muscles, nerves, tendons, bursæ, and even integument are affected. Lesions of great importance are found in serous membranes, in the heart, kidneys, reticulo-endothelial structures, intestines, and in other locations.

Articular lesions are considered first in this chapter. Subsequently, alterations of muscles, nerves, tendons, bursæ and integument are reviewed. Visceral lesions are discussed in another chapter.

[illegible]

Klinge (1934),[23] and Collins (1949).[84]

Although work in this field has gone ahead rapidly in recent years, much still remains to be done and some aspects of the pathology of rheumatoid arthritis have been studied in only a limited manner. Contributing factors responsible in part at least for the relatively slow unfolding of this field of medical research are the mechanical and technical difficulties connected with obtaining proper specimens from articulations and difficulties in preparing bone-containing specimens for pathologic study.

THE ARTICULAR LESIONS

Characteristics of articular lesions in rheumatoid arthritis depend largely upon the structural features of affected joints Freely movable joints show quite different changes from those which occur in less mobile joints. Understanding of the pathologic articular lesions in rheumatoid arthritis is therefore facilitated by knowledge of the gross and microscopic anatomy of various types of articulations. This subject will therefore be reviewed herein briefly

Joints which permit free movement are designated *diarthroses*, and it is among diarthroses of extremities that the most characteristic lesions of rheumatoid arthritis are to be found. Joints which permit little or no movement are designated *synarthroses*. The sacroiliac joints are notable examples of synarthroses and these, too, are frequently affected by rheumatoid arthritis Among synarthroses, three subvarieties have been described by anatomists, each being distinguished by the nature of its connecting elements. Where connecting substance is bone, articulations are classed as

PERIODICALS OF INTEREST TO RHEUMATOLOGISTS INCLUDE THE FOLLOWING

1. Annals of Rheumatic Diseases
2. Archives of Physical Medicine
3. British Journal of Physical Medicine
4. Journal of Bone and Joint Surgery
5. Occupational Therapy and Rehabilitation.
6. Physiotherapy Review.
7. Revue d'Orthopédie
8. Rheumatism and Arthritis (Rheumatism Reviews) (appearing in Annals of Internal Medicine).

BIBLIOGRAPHY

1. Arthritis Clinic Manual—Prepared by the Arthritis and Rheumatism Foundation, New York, 1952.
2. HENCH, P S and BOLAND, E W Ann Int Med, 24, 808, 1946
3. Primer on Rheumatic Diseases Jour Am Med Assn, 139, 1068, 1949

synostoses; if cartilage connects adjoining bones, the joints are classed as *synchondroses*; and if connective tissue intervenes, the designation is *syndesmosis* Synostoses will not figure in the discussions which follow as these are not affected in rheumatoid arthritis, probably because the synostoses are already solidly fused when rheumatoid arthritis makes its appearance.

Synchondroses and syndesmoses are usually attacked in rheumatoid arthritis Lesions found in the latter classes of joints are discussed below in a section on pathology of rheumatoid spondylitis

Movable diarthrodial joints and slightly movable varieties of synarthroses are closely allied from a developmental point of view Both

rounded by dense fibrous tissues In the case of true joints, or diarthroses, articular cavities increase greatly in size, become lined with flattened mesodermal types of cells and later become invested with capsular structures

Layers of flattened, plate-like cells lining articular cavities do not provide universal surface coverings; in some regions loose fibrous-tissue stroma of synovium is exposed.

Articular surfaces of bones are covered with hyaline cartilage Synovial membrane normally extends to the base of such cartilage, the chondro-osseous junction, fusing at this point with a fibrous membrane designated the perichondrium

Articular cartilages are devoid of blood vessels, hence do not react to inflammatory stimuli as do other mesodermal tissues Closely adherent to under surfaces of cartilages are the subchondral bone-plates, distinguished from other bone by compactness and absence of an Haversian system

Articular capsules are composed of two layers, an external layer of dense fibrous tissue and an inner or synovial layer, more cellular and having a stroma of lesser density. The synovial layer extends beyond the fibrous capsule covering all structures projecting into joints with the exception of

vessels

Synovial membranes may be thrown into temporary folds which appear only upon movements of the capsule In addition, permanent villi are often present, projecting into the articular cavity Villi may be pedunculated, with long thin stalks, or sessile, with short stocky trunks. In the adult some villi contain islands of cartilage.

Nerve fibrils which accompany blood vessels are known to arborize, ... A few Paccinian ... ly constitutes the

Diarthrodial joints can be examined at necropsy readily whereas more difficulty is experienced in obtaining material from synarthroses.

Pathologic changes of rheumatoid arthritis appearing in diarthrodial joints are progressive in character, possessing a dynamism which is in keeping with the varied clinical pictures presented by living patients. Determining variables responsible for the changing pictures include factors such as the patient's age, duration of arthritis, associated presence of osteoarthritis from unrelated causes, degree of activity of the rheumatoid arthritis (whether progressive or inactive) and character of previous treatment, whether indifferent or vigorous

Rheumatoid arthritis attacks each of the constituent structures of diarthrodial joints; consequently, it is convenient to consider the pathology of rheumatoid arthritis in terms of alterations which take place in encapsulating membranes, cartilage and epiphyseal bone.

Changes in Encapsulating Membranes.[1, 2, 12, 14, 19, 21, 22, 23]—Gross examination of synovial linings in rheumatoid arthritis generally discloses significant disturbances. The surface usually is reddened and the membrane thickened by edema. In late stages, the synovial lining may be greatly hypertrophied, appearing to bulge out of an incision made into the capsule. Some membranes are smooth and glisten but more often the surface is rough and granular. In advanced cases the synovial lining is often marked by presence of many irregular papillomatous villi. Such hypertrophied villi are most prominent in the knee joint especially at the margins of cartilage and in the regions between condyles. Villi are usually greyish pink in color although some are deeply hemorrhagic.

The amount of synovial fluid is generally increased. In large joints, as in the knee, several ounces may accumulate Characteristics of this fluid are discussed elsewhere. (Chapter 4)

The external capsular layer may be visibly thickened and coarsened, especially in cases with arthritis of long standing. Pliability of such thickened capsular structures is greatly reduced thus accounting to some degree for limited motion.

A different interior appearance is seen in joints which have become the sites of extensive adhesions and fibrous ankyloses. In such cases interior membranes may be greyish-red in color. The cavity may be obliterated in part by firm fibrous adhesions some of which are strung out in threads, others may be formed into septa which reach from one surface to another. Extensive regions of the articular cavity may be obliterated in this manner.

The microscopic pathology of synovial membranes in rheumatoid arthritis is quite characteristic. Some variation is found in specimens from different joints and indeed in specimens from several regions of the same joint. However, the basic pattern may be considered pathognomonic of rheumatoid arthritis as it is not exactly duplicated in any other articular disease.

Fundamental features of this pattern are (a) hypertrophy, (b) vascularization, (c) cellular infiltration (d) accumulations of inflammatory cells in follicle-like collections, (e) pannus formation.

(*a*) *Hypertrophy.*—Hypertrophy affects several components of the synovial membrane. The lining layer of flattened endothelial cells is frequently reduplicated reaching eight or ten cell-thicknesses in depth at some points (Fig. 44*B*) Stroma is likewise often increased in amount, its consistency is variable, being delicately fibrillar in some cases, infiltrated

with fat cells in some, and completely obscured by a dense cellular infiltrate in others.

Hypertrophy affects
to become greatly enlar
cut in cross-section, ap
structures. The internal structure of an inflamed villus is similar to that of synovium from which it arises. (Fig 45)

(b) *Vascularity.*—Synovia from rheumatoid arthritis show varying degrees of vascularity. Some are profusely supplied with small vessels, in others the vascular supply is modest. Vessels present are generally somewhat altered. Small arterioles (20 to 50 microns in diameter) may show

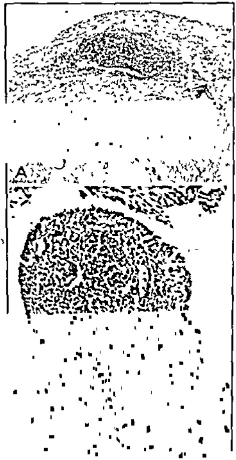


Fig 44 —*A* Rheumatoid arthritis Follicle-like collection of inflammatory cells in synovial
ar
to
he
of

rheumatoid arthritis indicating a disturbance of the nervous system Pigmentary disturbances, excessive sweating and Raynaud's like phenomena have also been considered evidences of nervous system disturbances

A study of peripheral nerves and sympathetic nerve chains from patients with rheumatoid arthritis, made recently, has contributed notably to information regarding pathology of this aspect of rheumatoid arthritis Characteristic inflammatory nodules of round or oval shape, sharply demarcated and resembling those described in voluntary muscles were seen distributed irregularly in nerves. These lesions were found not only adjacent to affected joints but also in places far removed from joints The nerve nodules could not be seen grossly in unstained specimens but were observed without the aid of magnification in slides stained with hematoxylin and eosin.

Neural nodules are now known to be located in the perineurium in most instances and are less commonly found in the epineurium None are found [illegible]

[illegible]

often found grouped in the same longitudinally-cut specimen Histologic-[illegible]

[illegible]

oid" type being polyhedral and having large elongated, irregular nuclei, poor in chromatin

Similar lesions have not been observed in control cases studied by Freund and [illegible]

to [illegible]

wor[illegible]

subcutaneous nodules, and to nodules found in synovia of rheumatoid arthritis. However, similar nodules have been observed in nerves of patients with rheumatic fever, dermatomyositis and lupus erythematosus [78] Therefore, as in the case of muscle nodules, the question of specificity does not appear to be settled at this time.

THE INTEGUMENT

Patients with rheumatoid arthritis are often found to have characteristic alterations of the skin. In cases with advanced articular destruction and deformities, severe atrophy develops in the skin and subcutaneous fat disappears. In such cases, the skin becomes withered and dry, desquamates freely and over distal portions of extremities may become tight and shiny. Patchy deposits of melanin pigment appear

In the presence of advanced and crippling deformities nails may become hooked or curved and are often thickened. In some cases their ends are turned up exposing a granular, bleeding nail bed.

Around the ends of fingers and particularly about margins of nails, skin may be darkly pigmented in some, in others bright red Similar redness

m[illegible]

Ir[illegible]

p[illegible]

thickened medial coats and often are surrounded by whorls of delicate fibrous stroma. Larger vessels may appear to have thickened walls Necrosis of arterial walls is not often observed and a regular tendency to perivascular cuffing is not observed. Dilated venous channels are usually present



Fig 45 — Rheumatoid arthritis Synovial membrane The interior surface is thrown into variously shaped villous folds, some have long pedicles, others are short and sessile All villi are hypertrophied The stroma is delicate and fibrillar. Hematoxylin and eosin × 70

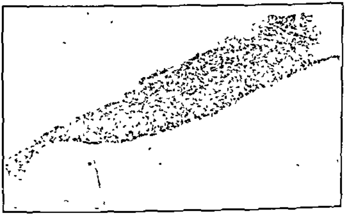


Fig 46.—Rheumatoid arthritis. Synovial pannus Cellular pannus extending over surface of articular cartilage Hematoxylin and eosin × 70

Histologically, superficial layers of skin show an apparent increase in the number of connective tissue cells but no evidence of severe inflammation.

CENTRAL NERVOUS SYSTEM

Histologic surveys of the central nervous system made recently disclosed no characteristic lesions attributable to the arthritis but evidence was found to suggest that certain lesions usually attributable to aging in the central nervous system are more pronounced and appear prematurely in persons with rheumatoid arthritis.[13] More advanced than in control cases were changes in anterior horn cells particularly in the lateral projections. These changes included chronic shrinkage, pigmentary atrophy and retrograde degeneration. Changes observed in the white matter included loss of myelin, disease and disappearance of axones and, hyperplasia of astrocytes The latter were not more advanced in arthritics than in control cases. Lesions were not encountered in upper motor neurones among the arthritic patients.

Electromyographic studies reported recently have shown spontaneous activity in muscles of patients with rheumatoid arthritis, suggesting that [illegible] Lesions [illegible] s the [illegible] with certainty.



FIG 50 —Hygromatous bursa (ganglion) from wrist Patient with rheumatoid arthritis Hypertrophy of villous lining with follicle-like collection of inflammatory cells resembling synovial membrane in rheumatoid arthritis. Hematoxylin and eosin. × 160

TENDONS, TENDON SHEATHS AND BURSÆ

Tendons, tendon sheaths and bursæ frequently show inflammatory changes in rheumatoid arthritis In acute and subacute cases, adjacent

(c) *Cellularity.*—Cells in the sub-endothelial layer are mainly lymphocytes, but always present are numbers of plasma cells, also reticuloendothelial cells, polymorphonuclear leukocytes (of which some are eosinophilic), fibroblasts and occasional giant cells containing two or three nuclei make up the remainder. Cellularity is widely variable from case to case, being exceedingly dense in some and rather sparse in others Blood

cells are regularly present in synovial membranes of rheumatoid arthritis (Figs. 44A and 44B.) These have been observed for years, but have especially attracted attention since 1931 when Allison and Ghormley[1] wrote that these nodules constitute specific lesions of rheumatoid arthritis Others, have since considered these nodules non-specific reactions of synovia to any chronic inflammation but my own experience corresponds with that of Allison and Ghormley I have not found similar follicles in other types of human articular inflammation

synovial membrane Follicles consist mainly of lymphocytes and a lesser number of reticuloendothelial cells Some follicles have loosely arranged central portions suggesting a germinal center. Mitotic figures are occasionally found among the nuclei. Small blood vessels traverse some follicles but are absent from others. Ghormley and Deacon[10] found the stroma of rheumatoid follicles independent of that in adjoining blood vessels and concluded from this observation that follicles of rheumatoid arthritis are not perivascular accumulations of inflammatory cells

(e) *Pannus* —Pannus constitutes a most important phenomenon among the pathologic processes of rheumatoid arthritis, always playing an important rôle in determining the mechanical character of changes which result in this disease.

A pannus is a layer of connective tissue derived from the margin of synovial membrane where it joins with cartilage From this region, pannus extends over the surface of cartilage growing toward the interior of the articulation (Fig. 46) Grossly, pannus appears as a reddish, roughened tongue-like protrusion of tissue. Margins of pannus are generally irregular, in some instances being villous and shaggy. In the knee joint pannus may extend completely across anterior surfaces of condyles and may cover semilunar cartilages. Granulations derived from under surfaces of pannus may be directed into every crevice and opening in cartilage which it covers.

trating granulations derived from subchondral marrow. Between these two layers, intervening cartilage may be destroyed or absorbed. The cellularity of pannus is variable· in active cases intense cellularity is the rule, in quiescent cases pannus may be fibrous and relatively acellular. Exte-

tendons and tendon sheaths are sometimes tender and swollen. In chronic cases two types of bursal lesions are found, (a) small adventitious bursae

actions of articulations and bursæ.

Acutely occurring types of bursitis and tenosynovitis and chronic

synovial membrane are frequently present Interior linings of bursæ and tendon sheaths showing chronic inflammation are thrown into folds and villi comparable to those seen in synovial membranes



FIG 51 —Adventitial bursa from patient with rheumatoid arthritis Lining membrane consists of hyalinized collagenous tissue sparingly supplied with nuclei Hematoxylin and eosin × 235

Hygromatous bursæ of rheumatoid arthritis are generally connected with an adjoining articular cavity by means of a pedicle This structure is

sæ

nts

Adventitious bursæ are in all probability an accidental association with rheumatoid arthritis and result from chronic pressure over bony prominences An example often encountered is the bursa which appears over prominent bunions. Linings of such adventitious bursæ consist of connective tissue membranes made up of collagenous fibers and containing but few cells Cells present are mostly flattened fibroblasts some having long tenu-

sive destruction of affected joints results where pannus has been in existence for long periods.

Articular Cartilage.—The rôle played by cartilage in the pathologic changes of rheumatoid arthritis is a passive one. Alterations found result from several factors including destructive effects of ingrowing pannus, traumatizing effects resulting from use of damaged joints, and the inevit-



FIG. 47.—Rheumatoid arthritis. Subchondral region showing destructive effects of expanding granulations. Osseous lamellae are being absorbed and under surface of cartilage is being invaded. Hematoxylin and eosin. × 135.

... in keeping with the degree of advance-
racter
ment
Where
granu-
ice of

tilage
tissue. Beneath margins of damaged joints one occasionally ... newly forming osteoid tissue and islands where cartilage is being transformed into osseous tissue to constitute bony spurs.

Epiphyseal Bone.—Pathologic processes affecting epiphyses of long bones in rheumatoid arthritis include (a) osteoporosis, (b) appearance of granula-

ous fibers concentrically arranged. As a rule, a complete lining layer is not present although scattered numbers of flattened fibroblasts provide an incomplete lining. (Fig. 51)

Central cavities of adventitial bursæ contain mucoid material devoid of fibers and cells. This mucoid material infiltrates between fibers immediately adjacent to the interior cavity and, in consequence, this zone stains with mucicarmine stain.

A third type of "bursa" associated with rheumatoid arthritis is a cystic cavity resulting from fusion of multiple subcutaneous nodules. (Fig. 49*B*) The process by which this type of sac is formed resembles the process which takes place in tuberculosis when individual tubercles fuse to form larger fluid-containing cavities. Sacs of this type are occasionally found at the elbow or over a knee. In some instances, bursæ of this type reach considerable proportions, measuring up to 4 or 6 centimeters in diameter. Central cavities of such nodular bursæ contain yellowish necrotic débris resembling that found in caseous centers of all rheumatoid nodules.

BIBLIOGRAPHY

1 Allison, N and Ghormley, R K. Diagnosis in Joint Disease, Baltimore
2
3
4
5
June, 1947.
6 Clawson, B J. and Wetherby, Mac N. Amer Jour. Path, 8, 283-294, 1932
7 Collins, D H Jour. Path and Bact, 45, 97-115, 1937
8 —— Rheumatic Disease, 1, 38-45, 1939
8a Collins, D H Pathology of Articular and Spinal Diseases, Edward Arnold and Co, London, 1949
9. Curtis, A C, and Pollard, H M. Ann. Int Med, 13, 2265-2284, 1940
10 Dawson, M H Jour Exper Med, 57, 845-858, 1933
11 De Forest, G K, Bunting, H, and Kenney, W. E.. Amer. Jour Med, 2, 40-44, 1947.
12
13
14
15 Jour Lab
16
17
18 0-746, 1936
19 sogennanter
20 chronischer Gelenkrheumatismus, Stuttgart, F Enke, 1908
Jour Amer Med Assn 111 2451-2455, 1938
8
en der Knochen und Gelenke und
ed II in Handbuch der Specielle
inger, 1934
, 425-443 1932-1933

tions and follicle-like accumulations of lymphocytes in marrow spaces, nd dis-

an increased fragility of epiphyseal bone. In some severe instances an epiphysis may be crushed with pressure of the fingers Evidence of osteoporosis is seen microscopically by thinning of lamellæ, widening of marrow spaces and, appearance of increased amounts of fat in marrow. In some cases the subchondral plate takes a part in this process, becoming thin in some regions, or disappearing completely.



FIG 48 —Rheumatoid arthritis Subarticular erosion Detail of a segment from a cyst-like erosion beneath an articular margin The cavity of the cyst is occupied by granulation tissue poor in cells Hematoxylin and eosin × 75

(*b*) *Granulations* arising in marrow spaces of epiphyseal bone may play an important role in bringing about destruction of subchondral bone

marrow spaces show follicle-like accumulations of lymphocytes resembling those found in synovia Subchondral granulations may grow upward and ... from below (Fig 47) ... within subchondral

(*d*) Roentgenograms of ... arthritis commonly show *erosions* of various sizes. These are located usually near articular margins, or beneath subchondral plates. Histologically, such erosions are seen to be filled with granulations arising either from subchondral marrow, or from the under surfaces of synovial membranes. (Fig 48)

(e) The process of *eburnation or marbling*, as found in the subchondral plate generally is seen in regions where overlying cartilage has been destroyed. Eburnation is a transformation of normal bone into extremely compact bone having unusually dense mineral structure. The Haversian systems are preserved. Some Haversian canals in eburnated bone open directly onto the surface. Some are tiny, appearing narrower than normal, others are wide and seemingly dilated.

Lesions of Rheumatoid Spondylitis.—This subject requires special consideration. The articulations affected include not only numerous true, or diarthrodial joints but also many half-joints or synarthroses. The latter react somewhat differently to rheumatoid arthritis than do the diarthroses.

Information available regarding pathology of all phases of rheumatoid spondylitis is meager. Mostly, these data have been derived from a study of dried skeletons and there is reason to believe, therefore, that valuable data will yet be obtained from a systematic restudy of this subject

Lesions are found in many elements of affected vertebral columns; intervertebral discs, vertebral bodies, anterior and posterior longitudinal liga-

instances. The atlanto-occipital joint is affected not infrequently but the dens-epistrophic joint generally remains free.

Intervertebral discs show first a progressive atrophy of the annulus fibrosis, later gradual narrowing of intervertebral spaces. Through the degenerated *annulus grows osteoid tissue, later transformed into bone*. Thus the vertebral bodies are eventually bound together with strong bony bridges. Ossification of intervertebral ligaments does not occur. However, these may become calcified and, in such cases, partial or complete union of adjoining vertebral bodies results. In late cases, intervertebral ligaments of the entire column may be affected and calcification may appear in the other longitudinal ligaments also Vertebral columns showing advanced stages of this process give an appearance of being plastered over with a

ints and costovertebral joints

Changes which occur in these articulations are said to correspond completely with those affecting diarthrodial joints of the extremities. Klinge[23] *described accumulations* of lymphocytes and plasma cells in synovial membranes of apophyseal joints and noted also hyalinization of the fibrous capsule *together with degeneration* of cartilage Eventually complete ankylosis results in some apophyseal joints

There is a notable lack of information regarding details of pathologic lesions affecting sacroiliac joints in rheumatoid spondylitis although, characteristically, these are the sites of the first changes to be seen in roentgenograms. One early change is development of small bridges of granulations between the two surfaces. These later are converted to bone. Eventually a complete destruction of cartilages covering the sacral and iliac surfaces takes place and subarticular bone may become the site of sclerotic and destructive lesions.

In later stages, calcific changes may appear in periosteum of the ischium and pubis; destruction and ossification of the symphysis pubis takes place and ossification may extend into attached muscles.

CHAPTER 10

THE CLINICAL PICTURE OF RHEUMATOID ARTHRITIS

By CHARLES RAGAN, M.D.

THERE is no single pattern of development of the disease; a variety of clinical forms may be observed Although the onset is most common between the ages of twenty and forty-five, this is not limited to any age group and the disease may begin in infancy or senility. Women are affected much more frequently than men except in rheumatoid arthritis of the spine (where males outnumber females 10 to 1)[39]

Some cases of rheumatoid arthritis are preceded by an upper respiratory infection while in others, precipitating influences seem to include emotional stress, psychic or physical trauma and exposure to cold and dampness

Most cases of rheumatoid arthritis seen in civilian practice begin insidiously, although acute fulminating cases are seen occasionally.* For weeks or months before actual joint involvement becomes apparent, the patient may complain of tiredness, lack of pep, general malaise, fatigue, numbness and tingling in the extremities, gradual loss of weight, exhaustion, vasomotor disturbances and general debility. Sooner or later, one or more joints—most often the *proximal interphalangeal joints of the fingers—gradually become warm, painful and swollen*

Only one or a few joints may be involved in the early stages but eventually the symmetrical polyarticular distribution so typical of rheumatoid

reappear in a few weeks or months but in most instances, once the actual swelling has appeared, it remains or increases until the disease becomes arrested or quiescent.[26]

The *joint involvement* is *often distributed symmetrically* and there is a tendency for progression of the process from the peripheral joints toward the trunk. It is i[illegible]
are involved very [illegible]
the distal interpl[illegible]
presence of psoriasis of the nails. Recent pathologic studies have shown, however, that these latter joints are involved in some cases In order of *frequency of joint involvement in rheumatoid arthritis* are the proximal interphalangeal joints of the fingers, metacarpophalangeal joints, knees, wrists, feet, ankles, shoulders, elbows and hips *Any joint in the body may become involved* in the process.

* In the military service, however, the reverse is true. A majority of cases of rheumatoid arthritis seen in the Army Rheumatism Centers during the past war had an acute or subacute onset. (Ed)

PATHOLOGY OF SUBCUTANEOUS NODULES

Subcutaneous nodules, highly characteristic lesions of rheumatoid arthritis, are found in 10 to 20 per cent of cases Usually such nodules are located over regions where concomitant factors of pressure and trauma impinge on bony prominences. Their duration is occasionally brief (smaller nodules may come and go within days or weeks) but nodules usually persist



FIG 49 —*A*. Rheumatoid arthritis Subcutaneous nodule Central zone of necrosis showing cellular and fibrous elements undergoing dissolution Surrounding layer of palisaded cells is incomplete Hematoxylin and eosin × 240 *B* Subcutaneous nodule Low magnification (× 14) showing numerous foci of necrosis in larger nodule Liquefaction has resulted in appearance of large clefts having irregular, branching cavities Hematoxylin and eosin

for longer periods and when firmly established persist permanently. Some are attached to fibrous external portions of articular capsules, some are incorporated into periosteum, some lie in loose subcutaneous tissues and some are found in upper layers of the integument Histologic appearances of

In the average case, there is little systemic reaction other than a low grade fever and tachycardia with some loss of weight during the early stages of the process; however, in a small percentage of cases, one may see repeated mild (or occasionally severe) attacks of multiple joint involvement with fever, tachycardia, leukocytosis and swelling, redness and pain in the affected joints *In fulminating cases*, spiking temperatures up to 103° or 104° F., which may be maintained for weeks, have been seen. In some of these acute episodes, the white blood count may be elevated to 15,000 or 20,000 (although the white blood count is usually within normal limits in the majority of instances of rheumatoid arthritis—or even lowered in long-standing chronic cases). *It is extremely difficult in the occasional acute fulminating case to differentiate the disease from rheumatic fever in the early stages* [25]

Other features include muscular weakness and atrophy. In some instances, one may find subcutaneous nodules (as often as 20 per cent in

from 20 to

per cent);

Important laboratory findings include the *increased sedimentation rate* during the active stage of the disease and the *roentgen findings*, which must always be interpreted in the light of the articular distribution, severity and duration of the disease Other laboratory tests, except the blood count, urinalysis and Wassermann, are usually of little value to the general practitioner. Although the patient's serum agglutinates strains of hemolytic streptococci (Lancefield Group A) in about half of the cases, this test is difficult and valid results necessitate careful technique not possible in a routine diagnostic laboratory. The sera of a large number of patients with *rheumatoid arthritis has been found to agglutinate sheep cells, sensitized* with an antisheep cell serum, to a higher titre than unsensitized sheep cells [26] This has been termed the *differential sheep cell agglutination*. It mirrors the streptococcal agglutination reaction to a certain extent, but numerous exceptions are found. The antibody has been found to be in the gamma and beta globulin fraction of serum The test is infrequently positive in rheumatoid spondylitis, and in psoriatic arthritis False positive reactions are rarely, if ever, seen. (See also detailed discussion in Chapter 13) The negative findings in most instances in respect to antistreptolysin and antifibrinolysin titres are of academic interest only insofar as the practitioner is concerned.

anemia is often encountere

nodules requires an expert

at all, these from the nodules of rheumatic fever Synovial fluid findings are also of some value, at times, when the diagnosis is in doubt. (For details see chapters on Laboratory Findings, p. 186, Synovial Fluid, p 48 and Radiology, p 64)

Rheumatoid arthritis is a constitutional disease with symptoms affecting chiefly joints and muscles. The joint pathology is primarily a synovitis but sooner or later, there is involvement of the epiphyseal bone and bone marrow, cartilage, fibrous tissue of the joint capsule and the adjacent

rheumatoid nodules are distinctive and notably different from the nodules of rheumatic fever, gout and palindromic rheumatism.

Characteristic of the histologic structure are (*a*) agglomeration of smaller nodules into larger masses, (*b*) presence in the basic units of three layers; inner or central zone of necrotic tissue, surrounding zone of cells arranged with long axes at right angles to the zones of central necrosis, and enveloping stroma of granulating fibrous tissue, densely cellular in some cases and sclerotic in others (Fig. 49.)

The central necrotic zone is made up of disintegrated material, the consistency of which is caseous or liquid. It has been postulated that processes leading to necrosis of interiors of nodules consist first of "fibrinoid" swelling of ground substance of connective tissue fibers; later, disintegration of fibrils and nuclei develops Eventually all structures in the affected area disappear including blood vessels which become thrombosed, and later liquified From a starting nidus, necrosis spreads outwards into adjoining tissues, branching and flowing, eventually extending over a large area. Calcification occurs very rarely in the central necrotic zones

The cellular reaction immediately adjacent to central necrotic regions is characterized by a curious radial arrangement of cell axes. This layer is not always continuous, being entirely absent in some regions Cells thus arranged appear to be young fibroblasts, generally mononuclear but occasionally with two or three nuclei occupying a single cell Rarely, mitotic
s. Palisading occurs in other patho-
annulare and in some cardiac lesions

External to the palisaded layer is a stroma with varying cellularity, depending upon the age of the nodule. Numbers of lymphocytes, monocytes and other cells are found but polymorphonuclear leukocytes are not
Small

ing in both cellularity and vascularity. The stroma does not constitute a well-defined capsule, rather it blends into and fuses with adjacent fibrous structures

Although Clawson and Wetherby[6] found an indefinite type of streptococcus in 12 of 17 cases, careful search for bacteria and viruses has been fruitless in the hands of most other investigators, and inoculation of material from nodules into laboratory animals has failed to reproduce the lesion.[10]

Some similarities may be seen between nodules of rheumatoid arthritis
more im-
ence of
histo-
s to be
different clinical expressions of the same process Around the uniform "rheumatic" focus of fibrinoid swelling, he stated, there develops proliferation of mesenchymal cells. In rheumatic fever the resulting structure takes the form of the myocardial Aschoff body. In rheumatoid arthritis sub-
norphology than with
and also Bennett and
colleagues[3] have concluded that the two lesions represent expressions of a

muscles, so that in reality rheumatoid arthritis comprises synovitis, chondritis, osteitis, epiphyseal non-suppurative osteomyelitis and an associated localized or diffuse periarticular and intramuscular fibrositis.[24]

In typical rheumatoid arthritis, the *onset* is *usually insidious*, with *involvement first of the small joints of the hands*, with slow progression to larger, less peripheral symmetrical joints. These persons are often asthenic with marked vasomotor instability The joints are usually not red but are swollen and sore, with little heat.

ONSET AND PRODROMAL SYMPTOMS

The onset is insidious in most cases. Prodromes are usually present in most patients with rheumatoid arthritis and may take the form of fatigue, emotional upsets, transient articular or muscular pains, numbness and coldness of the extremities (dead fingers), nervous irritability, fatigue, loss of weight or low grade fever.[24] Some may notice the articular manifestation *without any preceding* symptoms.

Dawson noted that *in 8 per cent of his patients, the disease began as an acute polyarthritis* which simulated rheumatic fever. There are many variations in the onset and course of rheumatoid arthritis, and the diagnostic difficulties are often great. *In some cases, the onset and clinical picture resemble rheumatic fever very closely*, the persistence of the joint lesions and their resistance to treatment suggest rheumatoid arthritis However, it must be remembered that in adults, rheumatic fever can be protracted and may not respond completely to salicylates. The distinction between rheumatic fever and rheumatoid arthritis may be extremely difficult and the true nature of the disease is revealed only after the passage of considerable time.

Occasionally a single joint may be attacked from the onset and the spread [illegible]

who has been burning either the mental or physical candle at both ends may well be warned that rheumatoid arthritis is very likely in the offing, whether or not she has had intermittent joint swelling and discomfort." Fatigue and nervous irritability are frequent precursors of the disease. (see also chapter on Psychogenic Factors in Rheumatic Diseases).

Symptoms of Rheumatoid Arthritis
Sex incidence—*2 or 3 females to 1 male*[22]
Race—no particular predisposition
Location—more common in the temperate zones than in the tropics.
Climate—more frequent in cold, damp climate than in dry areas.
Season—symptoms begin most often in the spring and fall, during changeable weather.[21]
Type of patient affected—often thin, asthenic.

different disease process. These writers have stressed the fact that the

colleagues also noted that cellular collections, resembling myocardial Aschoff bodies, often found in nodules of rheumatic fever are not seen in rheumatoid arthritis. Vascularity is, in general, far more pronounced in rheumatic fever and polymorphonuclear invasion much greater.

fe

ap

generally appear insidiously without relation to articular exacerbations, and persist, often for many years

The significance of rheumatoid nodules is not understood, neither is satisfactory knowledge available to explain their pathogenesis One can only guess from the histologic appearances as to the mechanisms by which they arise. The progressing and extending regions of necrosis located in the central zones give the impression that an extremely toxic chemical agent has been released in the vicinity, an agent capable of destroying and dissolving various types of tissues About this zone of advancing necrosis, a limiting barrier is erected, the palisaded fibroblasts The surrounding stroma reacts to the resulting lesions as to a foreign body, attempting to wall it off with inflammatory granulations

STRIATED MUSCULATURE

In most instances of rheumatoid arthritis, the voluntary musculature shows early clinical evidence of involvement Muscle weakness, wasting, and spasms are found adjoining affected articulations. The degree of muscular atrophy is generally proportionate to the severity of the articular disease but in some instances reduction in muscle bulk seems disproportionate to other clinical features of this disease

The gross appearance of striated musculature is notable only for reduction in bulk and visible flabbiness, otherwise there is no recognizable change The histologic appearances are, however, notably altered and the changes which appear in the finer structure of voluntary muscles have attracted considerable attention in recent years Progressive atrophy of fibrils was observed by Marinesco (1924)[25] and in 1934 Klinge[23] described necrosis of muscle fibers and lymph-cell collections in muscles far from the

drome"

The problem of muscle lesions in rheumatoid arthritis has recently been made the subject of detailed study.[18,19] This work has shown conclusively that serial sections from muscle tissue obtained in cases of rheumatoid

Symptoms of Rheumatoid Arthritis—(*Continued*)
Age of onset—usually between twenty and forty, may occur at any age, but children comprise only 6 per cent of these patients [16]
Prodromes—anorexia, loss of weight; low grade fever, tachycardia, hypotension, *sweating of hands and feet, nervousness, chilliness, fatigue,* weakness, numbness, tingling, stinging and burning of fingers and toes,[4,21] which may feel "dead" (sometimes blue and sometimes white)
Onset—usually insidious, acute in less than 10 per cent of cases in adults (often acute in children)
Joint involvement—tendency to symmetrical involvement, with joint pains, swelling, stiffness and progressive muscular atrophy, asymmetrical joint involvement at the onset in 30 per cent [5]
Progression of joint involvement—often from peripheral joints toward the body, joints of the hands and feet are most often involved at the onset
Frequency of joint involvement—proximal interphalangeal (fingers), metacarpophalangeal, toes, wrist, knee, elbow, ankle, shoulder, hip, temporomandibular, sternoclavicular, etc [16]
Characteristic joint swelling—fusiform, spindle shaped swelling especially when the fingers are involved.
Pain—often fibrositic at the onset, aggravated by movement, not rapidly shifting from joint to joint, due to occurrence of muscular spasm and contractures in later stages.
Stiffness—usually present, especially after periods of rest, may become generalized
Fever—slight, low grade, may be marked (101° to 104° F) in exacerbations *(especially in children)*
Tachycardia—*usually present, even without fever, elevated out of proportion to the temperature*
Loss of weight—moderate to marked, from 10 to 50 per cent of the body weight may be lost in a few months [20]
Subcutaneous nodules—found in from 5 to 25 per cent of typical cases,[4,23] especially on dorsal surface of forearm 1 to 2 inches below the elbow joints, or over the spine or occiput in bedridden patients [16]
Muscular atrophy—marked in some cases, with or without muscular twitchings
Secondary anemia—frequent; of type secondary to chronic inflammation, which fails to respond to iron
Mental depression—marked in most cases
Skin changes—cold and clammy hands, smooth, shiny, atrophic appearance often noted in the extremities, skin over the palms and soles is often a deep pink color. In severe cases, bronzing of the skin over the face and extremities and patches of vitiligo are seen frequently. Association with psoriasis in approximately 5 per cent.
Generalized lymphadenopathy and slight splenomegaly occur in some cases.
The familial incidence of rheumatoid arthritis—it is not uncommon to find rheumatoid arthritis in siblings

ules contains a few reticulin fibers and a definitely increased amount of collagenous tissue.

Muscle nodules of [illegible] *cutaneous nodules by* [illegible] features Subcutaneo[illegible] two centimeters in dia[illegible] Muscle nodules remain minute and can be seen only with the aid of a microscope Nodules in muscles show an absence of central necrotic zones and do not display the palisading arrangement of cells frequently present in subcutaneous nodules.

Accompanying muscle nodules are regions where degenerative changes occur in muscle fibers These may show hydropic changes, edema, loss of [illegible] atrophy. [illegible] uscle lesion in rheumatoid arthritis is [illegible] no lesions whatsoever, others show densely occurring nodules Muscle nodules are not confined to muscles adjacent to affected joints but are found also in muscles far removed from joints

The question of specificity of these muscle lesions in rheumatoid arthritis has not been settled to the satisfaction of all who have studied this problem Some writers[15, 31] have been impressed with their distinctive character and consider them specific for rheumatoid arthritis. However, others have observed inflammatory collections of cells in voluntary muscles of persons suffering with a number of other diseases, examples being [illegible]

[illegible]

rheumatoid arthritis and, as in the case of the cellular nodules described above, arteritis was found in muscle tissues far removed from affected joints Characteristically, arteries involved showed involvement of all coats although in some lesions were confined to adventitial coats. Cells [illegible] und in granulo[illegible] us mononuclear [illegible] were noted in muscle fibers Integrity of endothelial lining cells was generally maintained and thromboses seldom observed. Vessels affected by these lesions ranged in size from 75 to 420 microns in diameter according to the workers who reported these observations. Thus, this lesion affected the medium-sized arterioles, sparing smaller vessels as well as larger, more muscular arteries

At present one can only conclude that widespread inflammatory lesions are found in the muscles of patients with rheumatoid arthritis, and al- [illegible]

PERIPHERAL NERVES

Neuritic pains, paresthesia, trophic disturbances of skin and nails, symmetrical involvement of joints and vasomotor disturbances are features of

rheumatism described by Hench,[25] symptoms of muscular stiffness, or pain *in periarticular structures, usually labelled fibrositis and myositis*, may persist for years before *persistent joint changes* permit one to reach a diagnosis of rheumatoid arthritis. Included in these precursors of rheumatoid arthritis by some are chronic monarticular synovitis and intermittent hydrarthrosis.

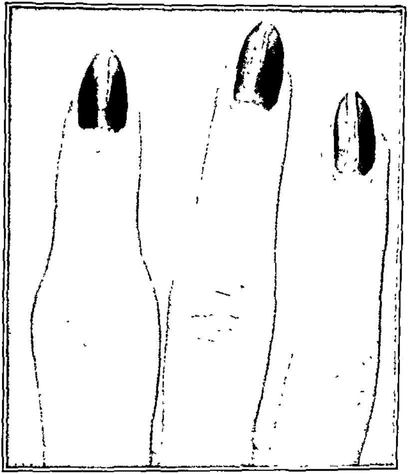


FIG 52 —Second, third and fourth fingers in a young woman with *rheumatoid arthritis*; this illustrates the marked fusiform (spindle shaped) swelling which often occurs in the proximal interphalangeal joints. It is seen best in the finger on the extreme left

The early course of rheumatoid arthritis is characterized by remissions and relapses In the earliest stage of rheumatoid arthritis, the patient may [illegible] ge joints [illegible] At this [illegible] the pres-

... normal in

rheumatoid arthritis. At the beginning of the disease, there may be few constitutional symptoms.[24] Persistent stiffness after inactivity, most marked on arising in the morning, may precede any joint symptoms. The stiffness may then be followed by joint pains which are migratory and leave no residual change. The joints first affected are most commonly the proximal interphalangeal and the metacarpophalangeal joints. Motion of the affected joints results in pain.

As the disease progresses, *joint pain* and *swelling increase* and *muscular* ... otective splinting about a painful joint ... in a diarthrodial joint, one group of ... the flexors—flexion contractures may develop if precautions are not taken. The sedimentation rate becomes definitely elevated, and the *patient gradually loses weight, becomes anemic* and has a poor appetite. With progression of the arthritis, the definite *fusiform swelling* makes its appearance about the joints affected (particu-

nounced because of muscular atrophy proximal and distal to the joint. The hands are cold and clammy and the skin covering these parts becomes atrophied, smooth and shiny.

The *constitutional symptoms* become more marked, with fever, tachycardia, sweating and further loss of weight and appetite. The patient becomes somewhat incapacitated.

In the *advanced stage* of rheumatoid arthritis, all of the constitutional symptoms become more marked and some of the affected joints show definite *flexion contractures* even under the best of care. These may be minimized with the proper application of splints. Partial or complete *ankylosis* may have occurred in some joints. If the disease continues, it may eventually burn itself out so that the sedimentation rate slowly returns toward

various joints.[24]

Fever.—*A low grade febrile reaction and tachycardia are usually present.* Some patients with rheumatoid arthritis may have no fever.[12] Although the temperature usually remains below 100° F., a recurring fever as high as 104° F. (without other obvious cause) has been reported, especially in children. *Low grade fever may persist for months* and may recur following an intermission. Fever may not be noted by the patient, in some individuals, the febrile period can be predicted by the gradual appearance of a

months, may be encountered. Tachycardia is frequently present in

nonarthritic controls.[1] The incidence of "various forms" of arthritis in ... O'Leary[10] as 12 per cent, by Brun-

... presents merely

... matoid arthritis

possibly distinctive in type.

Those who are reluctant to accept psoriatic arthritis as a specific entity point out that:

other joints. The unusual destructive lesions, sometimes found and attributed to the combined diseases may represent a rare form of rheumatoid arthritis [1]

The arthritis may occasionally precede the onset of the skin lesion or may appear almost simultaneously with it, but the joint disease is most often

In 20 patients observed by Dawson[4] the psoriasis preceded the arthritis in 15 In 9 of the patients, exacerbations and remissions of the arthritis accompanied similar changes in the psoriasis

The activity of the disease and ultimate deformity have been reported to be less in psoriatic arthritis than in simple rheumatoid arthritis but some patients may develop marked ankylosis and deformity. Psoriasis like rheumatoid arthritis often disappears during pregnancy.[8]

Dawson[4] has reported 19 cases of psoriasis with rheumatoid arthritis; in

rheumatoid arthritis, even in the absence of fever; it may continue for months after evidence of joint activity has disappeared.

MISCELLANEOUS FINDINGS

Loss of Weight.—From 10 to 50 per cent of the body weight may be lost in a few months. Some individuals may lose 100 pounds during the course of six months. In more than 50 per cent of these patients, a moderate loss of weight occurs (from 10 to 20 per cent of body weight) unless the caloric intake can be maintained at a high level. A gain in weight usually precedes *clinical improvement*.

Subcutaneous Nodules.—Subcutaneous nodules may appear in from 5 to 25 per cent of patients with rheumatoid arthritis.[14,15,26] This percentage undoubtedly varies tremendously in various areas. Such nodules are usually larger and persist longer than the corresponding lesions of rheumatic fever. *Dawson has found them to vary in size from tiny seedlike particles to small walnuts.* These nodules may persist for months or years. Millet seed nodules have been described by Vincent Coates of Bath. More than 100 nodules of different sizes have been observed in a single patient.[19] They may be attached to tendon sheaths or bursal walls but the skin overlying them is usually freely movable. Traumatic influences probably determine the sites of their occurrence; continued trauma may result in increase in size of the nodule to such an extent that it may resemble a bursa.

The commonest site for subcutaneous nodules is the extensor surface of the forearm about 1 or 2 inches below the elbow joint. They may also occur in the olecranon bursa, over the patella, scapula, spinous processes, sacrum, scalp, along the tendons of the fingers and toes, and in the eye (scleromalacia perforans).[4] These nodules are painless unless exposed to marked pressure because of their positions. We have never seen a microscopically proven nodule develop in a patient with rheumatoid arthritis who had a negative agglutination test for Group A hemolytic streptococci. *The presence of such nodules is an indication of a poor prognosis.*

The histologic similarity of the subcutaneous nodules of rheumatic fever and rheumatoid arthritis has been discussed by various writers,[9,14,19] while others[7] have pointed out dissimilarities between the nodules. In an excellent study of large numbers of subcutaneous nodules removed from patients with rheumatoid arthritis and from those with rheumatic fever over a period of ten years, it was found that the nodules in both diseases were similar in that each consisted of a central zone of necrosis, an intermediate zone of proliferating cellular tissue and a peripheral zone of inflammatory tissue.[7] However, in the rheumatic fever nodules the central zones showed predominantly a "fibrinoid" change as contrasted with a predominance of degeneration and necrosis in those of patients with rheumatoid arthritis.

The "fibrinoid" change in the rheumatic fever nodules was characterized by swollen, hyalinized, widely separated collagen bundles with persistence

The principal changes seen in the nails adjacent to affected joints are pitting, change in color of the nail plate, thickening of the nail plate, longitudinal ridging and erosion.

Etiology.—The etiology of the arthritis remains controversial. Two views continue to prevail:[15] (1) that the skin lesions produce a toxin which

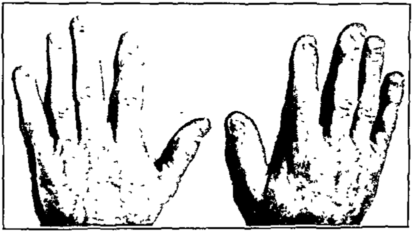


Fig 59 —Psoriasis of nails and arthritis of distal joints of fingers (early)

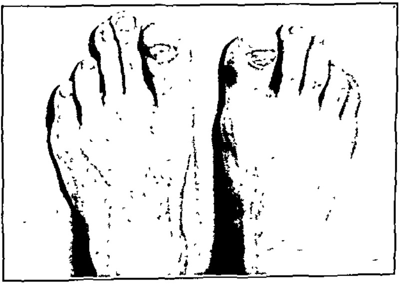


Fig 60 —Psoriatic arthritis in toes (same patient as Fig 59).

... of the arthritis The ... f the skin and nails ... us result from some agent acting on the ... d are therefore co-

The nodules of rheumatoid arthritis revealed a predominance of marked proliferation and degeneration of connective tissue with a complete absence of Aschoff-like nodules; exudation was not prominent in these lesions

stains metachromatically with toluidine blue and takes the Hale stain in the manner of an acid mucopolysaccharide

It was concluded that inasmuch as the rheumatic fever nodules are chiefly an exudative process, they may appear and resolve very quickly, leaving little residuum. The longer duration and larger average size of the nodules

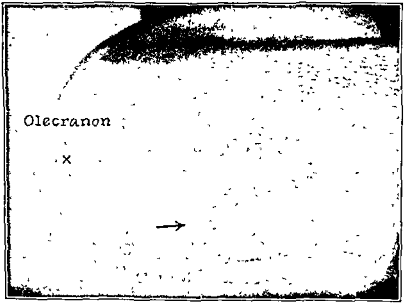


FIG 53 —Subcutaneous nodule on extensor surface of forearm below the elbow joint

—with warm moist hands In some patients there is a characteristic redness of the thenar and hypothenar eminences [26] *Bronzing of the skin* over the face and extremities has been noted in some of the severe cases Vitiliginous patches have also been observed.

Psoriasis and rheumatoid arthritis are discussed in a separate section.

changes Some have been reported[1,16] in which such marked atrophy and destruction occurred in the phalanges of the hands and feet that some of the phalanges were almost completely absorbed. In some instances the destructive process extended to the metacarpals and carpals.[8] Bony involvement in the proximal portions of the extremities is not as common but Jungmann and Stern[9] have reported a possible case of psoriatic arthritis of twenty years duration in which the psoriasis and joint trouble commenced simultaneously. Roentgenograms showed partial or complete dissolution of certain carpals, metacarpals, phalanges and humeral heads, tapering of the ulnæ, metacarpals, clavicles and metatarsals; destruction and dislocation of the sternoclavicular, acromioclavicular and shoulder joints, fusion of the radiocarpal joint and changes in the spine, suspicious of ankylosing spondylitis Such extensive bone and joint changes with loss of bony substance, tapered narrowing of bony fragments and "concertina-like" appearance of the folded skin over the involved joints has been described rarely in patients in whom no psoriasis was present. Four of such cases have been reported under the significant name of "Opera Glass Hand" or La Main en Lorgnette [17] (See also page 99).

Bauer and his associates[1] observed 26 patients with chronic arthritis and

have frequently observed, clinically and at autopsy, involvement of the terminal interphalangeal joints in patients with ordinary rheumatoid arthritis without psoriasis. The lesions of the terminal joints in these patients were similar to those occurring elsewhere, but differed markedly in one psoriatic patient with arthritis limited to the terminal interphalangeal joints [1]

were also present Ankylosis, subluxation with deformities and atrophy of bone were found in the advanced cases.

However, a striking difference was observed in the one patient with arthritis limited to the terminal phalangeal joints. This patient first noted arthritis in the terminal joint of the left index finger at twenty-five years of age, and subsequently developed deformities in most of the terminal joints of the other fingers and thumbs during the next forty-four years; these were associated with changes in the nails and paronychial inflammation. Psoriasis was first noted at the age of thirty-nine, fifteen years after the first joint involvement During the last twenty-two years of his life he had also had arthritis in most of the distal joints of the toes. The shortened fingers and toes, and swollen and deformed terminal joints were readily

In chronic rheumatoid arthritis, the *nails may become dry, brittle*, and may *develop longitudinal striations of* the nail plate with or without the formation of fissures (onychorrhexis) Subungual hyperkeratosis may appear and in some instances, there is a gradual separation of the nail plate from its bed, beginning at the free edge and progressing slowly toward the root (onycholysis); the entire nail may be lost In some longstanding cases, the nails of the toes may become elongated, raised, thickened, green or black in

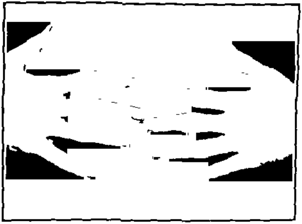


FIG 54.—Typical swelling and spindling in rheumatoid arthritis (Young, Jour. Bone and Joint Surg)

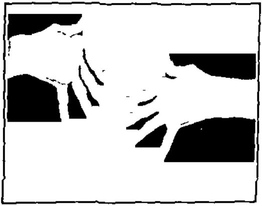


FIG 55 —*Ulnar deviation and deformity in rheumatoid arthritis* (Young, Jour Bone and Joint Surg)

color and may become twisted like a hook or spiral with an irregular and opaque surface; it may resemble a claw or a horn, and the texture of the nail resembles very much that of the horn of an animal (onychogryphosis) Pitting of the nails has been observed in some cases of rheumatic fever.[33]

PROGRESS OF THE DISEASE

The initial pain of rheumatoid arthritis is often generalized and fibrositic in nature, later becoming localized to the joints *The severity of the pain is*

apparent clinically. At autopsy following a coronary artery occlusion, the joints of both feet and one hand were examined minutely.[1]

Marked articular destruction and resorption of bone, with pronounced shortening of the middle phalanges, had occurred in the terminal phalangeal joints of the fingers and toes In the distal phalanges, the resorptive [illegible]

[illegible] toid arthritis was not present Dense acellular fibrous tissue (with minimal or no inflammatory changes) replaced the joint spaces in most of the digits There was no involvement of the proximal interphalangeal, metacarpophalangeal or metatarsophalangeal joints (which are usually affected in typical rheumatoid arthritis).



FIG 61 —Psoriatic arthritis (Shlionsky and Blake, courtesy of Ann Int Med)

Bauer concluded that in most instances the joint changes in rheumatoid arthritis are indistinguishable from those in rheumatoid arthritis associated with psoriasis. However, in an occasional patient unusual and destructive alterations may occur sufficient to suggest important differences in pathogenesis. It is Bauer's belief that if the term psoriatic arthritis is to be used, it should be restricted to cases in which the arthritis is limited to the terminal phalangeal joints.

Treatment.—The treatment of most cases of arthritis associated with psoriasis is similar to that described in the General Outline of Therapy for Rheumatoid Arthritis (p. 224) The various measures advocated for treatment of the systemic and local lesions should be included in the therapeutic program

Gold salts are not contra-indicated in the presence of psoriasis but such patients are often more sensitive, and toxic reactions occur more frequently

no index as to the ultimate prognosis. Although joint pain is usually relieved by rest and aggravated by motion, the muscular stiffness is at its worst after the patient has been inactive in bed. In the early stages, migratory joint phenomena may render the distinction from rheumatic fever difficult However, the pains do not shift from joint to joint as rapidly as in rheuma-

tension.

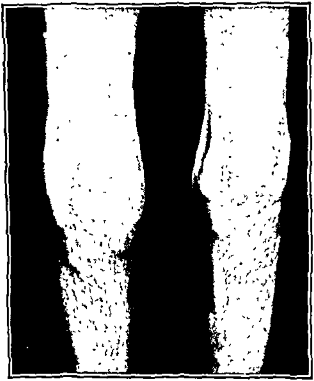


Fig 56.—Rheumatoid arthritis of the knees in a middle aged male

Monarticular involvement is unusual. *Any joint of the body may be involved* The joints first affected in most cases are the hands, especially the proximal interphalangeal and the metacarpophalangeal joints of the ring fingers Severe involvement of the wrists may also appear at an early stage, followed by arthritis of the knees and ankles, the hips are affected infrequently At the beginning and again later in the disease, stiffness of the neck and jaw may appear. Marked destructive changes in the jaw are uncommon. Following a period of prodromal joint pains and stiffness, the joints may become permanently involved and the process passes into a subacute or chronic form.

than in the ordinary group of rheumatoid arthritis. If the use of gold is contemplated it is advisable to proceed with more than usual caution and

inal joints and adjacent
be beneficial It is be-
y stages can be largely



FIG 62 —Psoriatic arthritis (Shlionsky and Blake, courtesy of Ann. Int Med)

controlled by vigorous therapy to control the psoriasis [13] If this is true, treatment of the skin is an important feature in the therapy of psoriatic arthritis Many favour the Goeckerman[8] regimen for patients with extensive skin lesions The treatment as outlined by O'Leary[10] consists of the use of coal tar ointment, ultra-violet light, oatmeal baths and autohemotherapy, but according to O'Leary the use of ointments is of no value in the treatment of psoriasis of the nails Perlman[11] claimed that undecylenic acid administration in arthritis with psoriasis was beneficial to both conditions In twelve patients, suffering from psoriasis with rheumatoid arthritis, Hollander and Lansbury[4] observed no objective improvement from large oral doses of undecylenic acid

Pain, stiffness, limitation of motion and swelling are the main symptoms noted by the patient Pain associated with changes in the weather is complained of by almost every patient but no relationship between barometric pressure or temperature fluctuation and pain can be charted accurately. This association of pain with the weather has no adequate explana-

hood of involved joints. The normal contour of the joints is restored only in exceptional instances following a subsidence of activity in this disease.

The Course of Rheumatoid Arthritis.—The course usually runs over a period of many years. *The progress of the disease may be interrupted temporarily or permanently at any stage so that no patient with rheumatoid arthritis should be considered as beyond aid.* On the other hand, *cure must not be promised in rheumatoid arthritis inasmuch as most individuals, even though markedly improved, will still manifest some degree of joint involvement from time to time.*

Accurate statistics on the natural course of rheumatoid arthritis are not available Short and Bauer[39] have reported on the course of 250 patients with rheumatoid arthritis receiving simple medical and orthopedic measures over a period of years. In 80 per cent, the observation extended over five years. Fifteen per cent were in remission, 38 per cent were somewhat improved, 13 per cent were the same and 34 per cent were worse. Of the patients who had had the disease for more than one year, only 5 per cent were in remission, whereas 56 per cent were stationary or worse These statistics are open to the criticism that some of the patients seen early in their disease may have had adult rheumatic fever. We feel that this cannot be distinguished clinically from rheumatoid arthritis In the same report, it was stressed that symmetrical, extensive, severe disease gave a poor prognosis. Factors which were of *no* importance in ultimate prognosis included prodromal symptoms, a progressive course before admission, the acuity of the onset, the presence of anemia, a family history of rheumatoid arthritis or rheumatic fever, or an onset associated with the menopause The males did slightly better than the females. These authors also stressed the intermittent nature of the disease since almost half of those who had improved within two years later relapsed The experience of a large group of rheumatologists has shown that somewhat less than 25 per cent "re-

One of the most pronounced general features of rheumatoid arthritis is the *muscular atrophy*, although the general nutrition of these individuals suffers severely. There is usually involvement to a greater or less degree of the connective tissue structures throughout the body and profound ca-

... this.[25] All of the

...ular atrophy is

The effect of Cortisone and ACTH on psoriatic arthritis is similar to that seen in rheumatoid arthritis.[12] A prompt antirheumatic effect follows the use of both drugs. Pain, swelling and tenderness are relieved while the drugs are administered, but in the patients thus far reported, relapse has occurred promptly when the hormones are discontinued. The effect on the psoriasis has been more variable and disappointing. Some patients have shown partial clearance of the cutaneous lesions, but others have developed new lesions during the administration of the hormone.

BIBLIOGRAPHY

1 Bauer, W, Bennett, G A., and Zeller, J. W.: Trans Assn Am Phys, 56, 349, 1941
2 Bourdillon, C Psoriasis et orthopathies, Thèse de Paris, No 328, 1888
3 Brunsting, L A Jour Michigan Med Soc., 42, 546, 1943.
4 Dawson, M H Med Clin. North America, 21, 1807, 1937
5 Epstein, E. Arch Dermat. and Syph, 40, 547, 1939
6. Goeckerman, W H: Northwest Med., 24, 229, 1925
7 Hench, P. S · Ann Int Med, 11, 1089, 1938
8 Hollander, J. L, and Lansbury, J · Personal Communication
9 Jungmann, H, and Stern, V S. Brit Jour. Radiol, 17, 383, 1944
10 O'Leary, P A Canad Med. Assoc. Jour, 48, 34, 1943
11 Perlman, H H: Jour Am. Med Assn 139, 444, 1949
12. Polley, H. F, Hench, P S, Brunsting, L. A Jour. Lab. and Clin. Med, 36, 973, 1950.
13 Rheumatism reviews Fourth, Ann Int Med, 11, 1089, 1938
14 ———. Eighth, Ann Int Med, 15, 1002, 1941
15 ——— Ninth, Ann. Int Med, 28, 309, 1948
16 Symposium on the practical management of psoriasis, Jour Infect. Dermat, 4, 399, 1941
17 Weigeldt, W. Münchner Med Woch, 76, 1270, 1929

tractures tend to develop as the periarticular swelling gradually subsides These *contractures*, unless properly attended to, result in the distressing deformities that are most apparent in the hands, knees, wrists and elbows In the hands, the usual deformities that develop, if the process is unchecked, include flexion of the proximal interphalangeal joints with atrophy of the interossei and thickening of the metacarpophalangeal and proximal interphalangeal joints Finally, ulnar deviation at the metacarpophalangeal

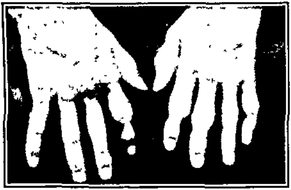


Fig 57 —Rheumatoid arthritis involvement of metacarpophalangeal and proximal interphalangeal joints

joints renders the hands almost useless Fibrous and/or bony ankylosis may occur together with subluxation or actual dislocation of these joints Another very distressing deformity which must be avoided is fixation of the knees in partial flexion [26]

of patients, the course of the disease is progressively downhill despite the therapy or the lack of the same. In the late stages of the disease, the characteristics presented by such patients are emaciation, muscular atrophy, joint deformity, contractures and ankylosis

* Muscle atrophy may also be caused by the focal inflammatory changes in muscles and nerve sheaths, found in cases of rheumatoid arthritis by Freund Steiner *et al* —See Chapter on Pathology of Rheumatoid Arthritis p 141 (Ed)

CHAPTER 12

THE VISCERAL LESIONS OF RHEUMATOID ARTHRITIS

By Edward F. Rosenberg, M.D.

It is not unusual to find in patients with rheumatoid arthritis, evidences of disturbed function or structure of organs other than joints. Resulting symptoms are so varied and in many instances so widespread that this disease must be considered a generalized ailment of which arthritis is but a single manifestation

Previous generations of physicians were not greatly impressed with the systemic character of rheumatoid arthritis. The younger Garrod, for example, an outstanding British rheumatologist of his time, stated in 1890 that visceral lesions which can be found at necropsy in patients with rheumatoid arthritis may be ascribed to intercurrent diseases and not to the articular disease[13] This, in general, represented the opinion of a majority of the physicians who studied this disease up to recent years. Researches of the last two decades have, however, shown numerous alterations both in the microscopic and gross appearances of various internal organs For many of these alterations, a relationship between visceral lesions and articular disease is presumed to be present. A description of the character

high percentage of the patients who are suffering with this disease

Typical of the findings in most of these studies as reported in 1941 by Baggenstoss and Rosenberg,[2] who noted that 16 among 30 patients examined at necropsy had "rheumatic" damage in the hearts Rheumatic lesions present in these individuals could not be ascribed to unrelated rheumatic fever as a history of acute rheumatic fever was obtained in

'run

nter

It was concluded that inflammatory heart disease and rheumatoid arthritis were in some manner related

The "rheumatic" lesions which were found in this study were often rather severe and extensive ones Subacute and chronic changes were noted in some, showing active inflammation or burned-out lesions without

Coss and Boots studied 56 cases,[13] and in their series the earliest age of onset was fourteen months. Thirty per cent had splenomegaly, 40 per cent had generalized glandular enlargement. The agglutination reaction with Group A hemolytic streptococci was found less frequently in juvenile rheumatoid arthritis than in adults. Colver[11] had felt that the duration of activity of the disease was never over 7 years but Coss and Boots were unable to corroborate this and mentioned one patient who had had periods of activity over a twenty-three year interval. Twenty-five per cent of their patients had a duration of seven years or more. *Brachydactylia* (shortened fingers from disturbed epiphyseal growth) was common.

Treatment of rheumatoid arthritis in children follows the same general pattern as in adults. Good results have been reported in a few cases following injection of gold compounds Angevine thought that more attention should be paid to the psychic factors associated with the home, school and

early activity than on prolonged rest in bed because of the importance of preventing muscular wasting. *Great stress must be placed on the prevention of deformities in these individuals.*

FELTY'S SYNDROME

Felty,[17] in 1924 reported a series of five patients with rheumatoid arthritis, marked leukopenia, and splenomegaly. While this term is still in use there seems little evidence that this syndrome is more than a minor variant of rheumatoid arthritis. The triad of rheumatoid arthritis, leukopenia, and splenomegaly may therefore be designated as Felty's syndrome, but the treatment is that of typical rheumatoid arthritis.

SJÖGREN'S DISEASE AND RHEUMATOID ARTHRITIS

Apparently another variant of the clinical picture of rheumatoid arthritis is its association with keratoconjunctivitis sicca (so-called Sjogren's Disease). At least three recent reports[27, 28, 35] emphasize the coexistence of the conditions, and express the conviction that the deficient lacrymal

s

sales

ting

eyes, dryness of the nasal, buccal, and pharyngeal mucosæ from reduction of salivary flow, and hoarseness.[28]

On physical examination the cornea of the eyes show punctate ulcerations, as demonstrated by rose bengal staining, the salivary glands are palpably enlarged, and joints show characteristic changes of rheumatoid arthritis in about 80 per cent of cases.[28] Also described as sometimes occurring with the condition are cough, achlorhydria, dryness of the skin,

-pecia, or even scleroderma-

n 62 patients with rheuma-

toid arthritis w

method used,

signs of activity. Degeneration of cardiac fibers, infiltrations with variou
types of cells, pericarditis, myocarditis, inflammation of coronary arteries
and in many instances typical Aschoff bodies were found (Fig. 63). In
many instances the valve leaflets were very severely affected; and in some
cases the inflammation present within valve leaflets extended beyond the
valve into the so-called "ring," the zone of tissue which supports the base
of valve leaflets.

In some instances lesions in the hearts are of a character which indicates
that their incidence occurred months or years prior to death and remain
in a state of continuing "activity." Pericarditis, both fibrous and exu-
dative (Fig. 64), occur in such instances; also, verrucous growths are
observed on endocardial surfaces of valves. In some it is possible for
pathologists to discern lesions which indicate rheumatic inflammation of
recent occurrence *superimposed upon older, inactive residues of previous*
episodes.

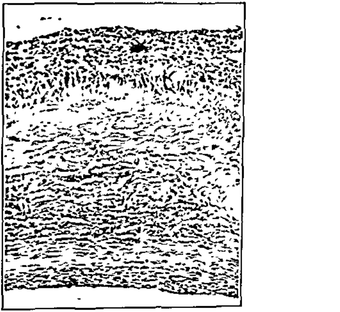


Fig 63 —Subacute aortic valvulitis Palisading of the basophilic cells around a region of fibrinoid degeneration Hematoxylin and eosin, × 200

In some hearts lesions are observed with histologic characters indicating a "burned-out" or inactive rheumatic inflammation. Blood vessels, newly formed, are found in valves and valve rings, deposits of calcium in valves and in the myocardium, peculiar scars which give the appearance of layers of onion skin

It is especially noteworthy that "rheumatic" heart damage in the rheumatoid arthritic patient is often severe and widespread, causing ex-
... Only rarely is it necessary to search in histologic speci-
... It is notable also that lesions found in
... "stigmata" of rheumatic fever which
...ery high percentage of the "run of the

Thus, they confirmed
decreased secretion of
umatoid arthritis, only
giving rise to the symptoms of Sjogren's disease when severe.

Treatment is the same as for classical rheumatoid arthritis. Sjógren's syndrome has responded well to ACTH and cortisone therapy.

BIBLIOGRAPHY

1 ANGEVINE, D M Clinics, 1, 582, 1942
2 ANGEVINE, D M, ROTHBARD, S, and CECIL, R L. Jour Am Med Assn, 115 2112, 1940
3 ATKINSON, F R B Brit Jour Dis Child, 36, 100, 1939
4 BAUER, W New England Jour Med, 221, 524, 1939
5 ———. Jour Am Med Assn, 115, 2024, 1940
6 ——— New England Jour Med, 229, 583, 1943
7 BENNETT, G A, ZELLER, J W, and BAUER, W Arch Path, 30, 70, 1940
8 BLOCK, W D, BUCHANAN, O H, and FREYBERG, R H Arch Int Med, 68, 18, 1941
9. COATES, V
10 COLLINS, D I
11 COLVER, T
12 COPEMAN, W York,

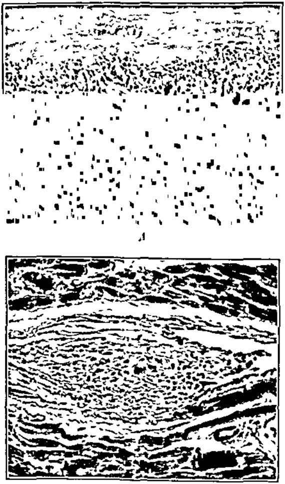
29, 143, 1946

nd Jour Med, 208, 1030, 1933.
itis, Nelson's Loose Leaf Med.,
In Press
17 FELTY, A R. Bull Johns Hopkins Hosp, 35, 16, 1924
18 HARTUNG, E F, and BRUGER, M Jour Lab and Clin Med, 20, 675, 1935
19 HAWTHORNE, C O Brit J Rheum, 1, 109, 1938
20 HENCH, P S Ann Rheum Dis, 2, 172, 1941
21 ——— Chronic Arthritis, *in* Barr's Modern Medical Therapy in General Practice, Baltimore, Williams & Wilkins Company, 3, 3298, 1940
22 HENCH, P S, *et al* Ann Int Med, 8, 1315, 1935
23 ———. Ann Int Med, 10, 754, 1936
24 HENCH, P S, OSGOOD, R B, and WAINWRIGHT, C W Army Med Bull No 60, January 1942

1936
34 PORTIS, R B Am J Dis Child., 55, 1000, 1938
35 READER, S R, WHYTE, H M, and ELMES, P C Ann Rheumat. Dis, 10, 288, 1951
36 ROSE, H M, RAGAN, C, PEARSE, E, and LIPMAN, M O Proc Soc Exper Biol and Med, 68, 1, 1948
37 SCHLESINGER, B Still's Disease, a Survey of Chronic Rheumatic Diseases,
38 S
39 S
40
41

mill'' cases studied at necropsy examinations. It has been stated that evidences of such stigmata can be found in as many as 90 per cent of such cases. Evidences of inflammation in the hearts of patients dying with rheumatoid arthritis are frequently widespread, causing serious deformities of the cardiac structures as to permit ready recognition on gross study of the affected organ.

Hypotheses which are currently considered as possible explanations of these findings are as follows. Perhaps rheumatic fever and rheumatoid arthritis are closely related or even identical diseases and, therefore, cardiac lesions produced in the one disease resemble those which appear in the



A

B

Fig. 61.—Chronic rheumatic lesions in hearts of patients with rheumatoid arthritis. (*A*) Rheumatic mitral valvulitis. Large basophilic and giant cells are arranged about a region of necrosis. Hematoxylin and eosin, × 195. (*B*) Myocardial Aschoff body. Hematoxylin and eosin, × 245.

CHAPTER 11

PSORIASIS AND RHEUMATOID ARTHRITIS

By WALLACE GRAHAM, M.D.

THE association of joint pains and psoriasis was noted as early as 1822[1] but the first comprehensive clinical study of these allied conditions was recorded by Bourdillon in 1888.[2]

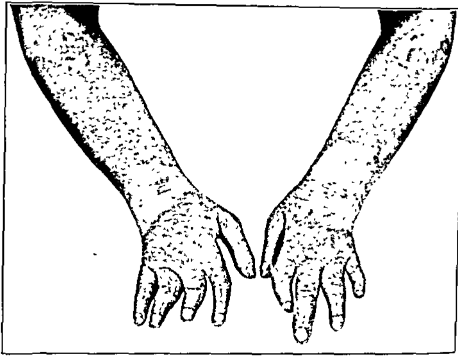


FIG 58 —Psoriasis and rheumatoid arthritis

Various names have been applied to the combination of rheumatoid arthritis and psoriasis; these include psoriatic arthritis, psoriasis arthropathica, arthropathia psoriatica, and psoriasis arthritica, but the application of these terms should be restricted as many cases merely represent psoriasis occurring coincidentally with unrelated joint disease For example, the occurrence of psoriasis with degenerative arthritis is considered to be merely a matter of chance.

Incidence.—*It is generally agreed that the association of arthritis of the rheumatoid type with psoriasis is significant and not merely the coincidental occurrence of two rather common diseases.*[15] Bauer and his associates found psoriasis in 2.7 per cent of 300 unselected patients with rheumatoid arthritis, but the disease was present in only 0 7 per cent of a similar number of

other. As matters stand, this hypothesis cannot be proved or disprove as the cause of both remains unknown. It [illegible]
whether

Also b
hearts of patients with rheumatoid arthritis may result from a differen agent than that which causes rheumatic fever. It may be that differen etiologic agents cause identical-appearing pathologic lesions in the hearts Should this hypothesis prove correct it will be necessary for many student of pathology to alter an opinion, now long-held, that morphologic lesion produced by rheumatic fever are "specific" and are not reproduced b any other ailment

There is a further possibility that patients with rheumatic heart diseas and rheumatoid arthritis may have both unrelated diseases At presen this seems most unlikely as in many instances no clinical data can be found to indicate that the patients had suffered independent episodes of rheumati fever

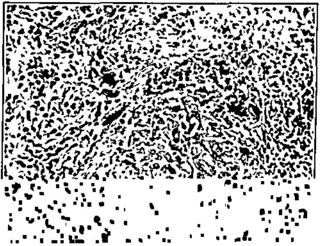


FIG 65 —Cellular nodule from the pericardium Hematoxylin and eosin, × 200

[illegible]

7 of 100 cases of rheumatoid arthritis and Monroe[19] found evidence of valvular damage (presumably rheumatic) in only 9 per cent of 267 patients with this disease. In one, detailed study[18] of the heart showed some electrocardiographic evidences of cardiac damage in 100 per cent of a series of cases of rheumatic fever but in contrast showed no heart damage whatsoever in patients with rheumatoid arthritis Another study showed evidences of enlarged heart or myocardial damage in six per cent of a series of patients with rheumatoid arthritis and signs of valvular defects in an additional three per cent of the same series.[20] This incidence was thought to be no more than that expected in the general population and was not considered to indicate affection of the heart by rheumatoid arthritis. A

high percentage of patients with rheumatoid arthritis in a Swedish hospital were found recently to have evidences of heart disease discovered by examinations which included electrocardiographic examinations during hypoxic states induced by having the patient inhale 9 per cent oxygen for

3.4 per cent, an incidence similar to that found among control patients in whom the incidence was 2 per cent.

It is apparent from these paragraphs that divergent results have been reported recently from researches conducted by pathologists on the one hand and by clinicians on the other It is certain, however, that a large number of patients with rheumatoid arthritis show seriously damaged hearts at necropsy



FIG 66



FIG. 67

FIG 66 —Aortic valve showing an extensive lesion in the leaflet and the ring Hematoxylin and eosin, × 8 A central zone of necrosis, peripheral inflammatory reaction and resulting large nodule formation are present This lesion resembles a subcutaneous nodule of rheumatoid arthritis

FIG 67 —Nodule in the mitral valve The zones of necrosis and peripheral inflammatory reaction resemble a subcutaneous nodule of rheumatoid arthritis

Unusual Cardiac Lesions Associated with Rheumatoid Arthritis.—In addition to lesions resembling rheumatic heart disease bizarre lesions, similar to those occurring in subcutaneous nodules of rheumatoid arthritis have been found in hearts of patients dying with this disease.[4, 11, 14] The predominant structural unit of this process is a granulomatous body, roughly spherical, possessing three well-demarcated zones; a central zone consisting of acidophilic, apparently necrotic tissue, a zone of palisaded reticuloendothelial cells having large pale-staining nuclei and faintly

Other Mortality Experiences.—Data concerning manner of death in 4 additional series have been reported during recent years In 3 series, pulmonary deaths led other causes; the lesion most often responsible being bronchopneumonia (24 cases in a series of 61 (39 per cent) studied by Fingerman and Andrus; 4 of 23 cases (17 per cent) reported by Bayles; 11 of 31 cases (35 per cent) reported by Young and Schwedel) Other fatal pulmonary lesions included pulmonary suppuration, pulmonary embolism, fat embolism, and so forth Bennett found pulmonary lesions second in frequency, 11 of 48 cases (23 per cent). These were exceeded by cardiac lesions as causes of death, among his patients.

Other causes of fatalities have been renal failure due to glomerulonephri- [illegible]

BIBLIOGRAPHY

1 Andrus, F C : Minnesota Med , 24, 1071–1072, 1941

2 Baggenstoss, A H. and Rosenberg, E F.: Arch Int Med , 67, 241–258, 1941

3 ——— Proc Staff Meet Mayo Clinic, 16, 232–240, 1941

4 ——— Arch Path , 35, 503–516, 1943

5 ——— Arch Path , 37, 54–60, 1944

6 Bayles, Theodore B Amer Jour Med Sci , 205, 42–48, 1943

7 Bennett, G A Ann Int Med , 19, 111–113, 1943

8 Clark, W S , and Bauer, W Ann Rheumat Dis , 7, 39–40, 1948

9 Dawson, M H , and Tyson, T L Jour Lab and Clin Med , 21, 575–587, 1936

10 Dixon, quoted by Kurnick [17]

11 Duke-Elder, W Stewart Textbook of Ophthalmology, Vol III, St Louis, C V Mosby Co , 1941

12 Fingerman, D L , and Andrus, F C Ann. Rheumat Dis , 3, 168–181, 1943

13 Garrod, A E A Treatise on Rheumatism and Rheumatoid Arthritis, Philadelphia, P Blakiston, Son and Company, 1890, pp 261–263

13a Goar, E L., and Smith, L S . Jour Am. Med Assn , 148, 889, 1952

14 Graef, I , Hickey, D V , and Altman, V : Cardiac Lesions in Rheumatoid Arthritis, Paper presented at the Inter-American Cardiological Congress, Chicago, June 13–17, 1948, to be published

15 Hall, F M , and Anderson, L R : Amer Heart Jour 25, 64–80, 1943

16 Helm, E *Trans Ophthalmol Soc of the United Kingdom*, 55, 478–482, 1935

17 Kurnick, N B Amer Jour Dis. Childhood, 63, 742–747, 1942

18 Master, A M and Jaffe, H L : Med Clin North America, 18, 759–769, 1934

19 Monroe, R T and Walcott, C F The incidence of cardiovascular disease in chronic arthritis Medical Papers, Christian Birthday Volume, Baltimore, Waverly Press, Inc , pp 918–927, 1936

20 Monroe, R T Chronic Arthritis, (in Oxford Medicine), New York, Oxford University Press, Vol 4, pt 2, pp 367–404, 1939

21 Rosenberg, E F , Baggenstoss, A. H , and Hench, P S : Ann Int. Med , 20, 903–919, 1944

22 [illegible]

23 [illegible]

24 [illegible]

25 [illegible]

26 [illegible]

27 [illegible]

basophilic cytoplasm and, peripheral to this palisaded layer, a broad and imperfectly delineated zone of inflammatory reaction. Sections stained for bacteria have failed to reveal any organisms. This lesion occurred in 2 cases among 34 reported by Baggenstoss and Rosenberg, in 2 of 66 cases studied by Graef, Hickey and Altman, and in 2 of 45 cases studied by Clark and Bauer. The total incidence was thus 6 cases among 145 (approximately 4.4 per cent).

The nature of this lesion is unknown. It may prove to be peculiar to and specific for rheumatoid arthritis, but it may also reflect merely an unusual lesion of rheumatic fever. (Figs. 66 and 67)

Non-rheumatic Heart Disease in Rheumatoid Arthritis.—Unrelated forms of heart disease have been encountered in many patients dying with rheumatoid arthritis. Among 30 cases studied by Baggenstoss and Rosenberg[2] 8 showed non-rheumatic lesions. Death resulted in 1 from coronary occlusion, and in a second from severe myocardial degeneration of an undetermined cause. In 6 patients, non-rheumatic cardiac lesions of minor severity were noted. These included (1) coronary sclerosis with chronic myocardial infarction, (2) hypertrophy of the heart resulting from hypertension, (3) hydropericardium; (4) nonspecific subacute pericarditis; (5) chronic obliterative pericarditis and (6) calcific aortic stenosis. Mild arteriosclerotic cardiac changes, consistent with the ages of affected patients and not severe enough in any instance to be fatal, were found in 13 of 22 cases studied by Bayles. Clark and Bauer noted atherosclerosis of the aorta and coronary arteries usually with degenerative changes of the myocardium "rather constantly" present among their patients.

TABLE 2—Postmortem Observations on the Lungs in Thirty Cases of Rheumatoid Arthritis[1]

	Cases
Fibrous adhesions between parietal and visceral pleura (22 cases)	
Associated with pericardial adhesions	10
Associated with healed tuberculosis of lungs and hilar nodes	7
Associated with healed tuberculosis and hilar nodes only	4
Associated with chronic active tuberculosis of lungs	3
Bronchopneumonia (9 cases)	
Responsible for death	3
Terminal	6
With empyema	2
With pulmonary abscess	1
With organization	2
Bronchiectasis (3 cases)	
Mild	1
Severe (Responsible for death)	2
Pulmonary embolism (4 cases)	
Fatal	3
Nonfatal	1
Pulmonary fat embolism	2
Emphysema	3
Bronchitis	1

CHAPTER 13

LABORATORY FINDINGS IN RHEUMATOID ARTHRITIS

By HOWARD C. COGGESHALL, M.D.

THERE is no laboratory procedure available which differentiates rheuma-toid arthritis from the other types of arthritides. No single test remain constantly abnormal throughout the course of rheumatoid arthritis. Th most significant information can be obtained from a determination of th erythrocyte sedimentation rate (see Chapter 5 p. 56), a complete bloo count and urinalysis. The roentgen examinations are helpful but frequentl disappointing by g[illegible] the moderately ac[illegible] should be applied [illegible] taken up elsewhere. Camp states: "The pathologic and roentgenograph response of joints in various diseases is too similar to permit dogmat interpretation of the roentgenogram."[14 55] The diagnosis depends mo upon the clinical history and physical examination than the laborator findings [19]

Blood Cytology.—Examination of the peripheral blood by counting th number of erythrocytes and leukocytes should always be done.[51 55] leukocytosis may exist early in the disease while later a normal or low cour may be encountered.[7 95] The white blood count is between 5,000 an 10,000 per cubic millimeters in 80 per cent of the cases; the count may b *from 12,000 to 20,000 in the acute stage* (Leukopenia is not uncommon i chronic long-standing diseases) In children, the leukocyte count ma [illegible]

estimating the severity of disease [illegible] There may be [illegible] number of polymorphonuclear *leukocytes using the routine* differenti leukocyte count, whereas the Arneth-Schilling technic may show an increa [illegible] forms of the polymorphonuclear leukocytes.[25 36 37 53 95–100] [illegible]

disease.[95] The Schilling count was found [illegible] group of 19 patients with active rheumatoid arthritis reported by Shor [illegible] and Bauer [95] They found it to be slightly less accurate than th [illegible]

pictur [illegible]

Among cases studied by Graef and his colleagues were 2 with lesions in the coronary arteries resembling those of periarteritis nodosa

Pulmonary Lesions.—Non-specific pulmonary lesions including a number of inflammatory and vascular diseases are frequently present in patients dying with rheumatoid arthritis. Adhesive pleuritis was found in 73 per cent of the cases in one series,[4] suggesting that episodes of pleurisy with subsequent healing occur frequently. Perhaps serous membranes as well as synovial membranes are attacked by the agent responsible for rheumatoid arthritis

As pleural adhesions are a frequent manifestation of tuberculosis, a possibility was considered that pleural adhesions resulted from tuberculosis in these cases. Careful search, however, revealed evidence of tuberculosis only in 14 of the 22 cases in which healed pleuritis was encountered. This would seem to indicate that the two conditions, pleurisy and tuberculosis, probably were not related in every instance studied.

The incidence of pulmonary tuberculosis in these cases is of considerable interest because rheumatoid arthritis has been suspected by some clinicians to be an aberrant form of tuberculosis. The incidence of pulmonary tuberculosis in this group (46 5 per cent) was notable, but not higher than in the general run of necropsies performed at the same institution. Active lesions were noted in only 3 of the 30 cases (10 per cent) in this series, and in 7 of 69 cases (10 per cent) of cases studied by Fingerman and Andrus [12]

Bronchopneumonia occurs in approximately a third of all patients who die with rheumatoid arthritis. This incidence is not unusual among patients dying with a serious chronic disease.

No evidence of so-called "rheumatic" pneumonia has been found in these cases

It was noted that fatal bronchopneumonia was precipitated in some cases by measures of therapy employed against the arthritis, for example, by [illegible]

[illegible] marrow spaces is released into the circulation [illegible]

[illegible] Despite the curious inter- [illegible]

[illegible] exceeded 1800 grams in eight of the cases The hypertrophy resulted from [illegible]

[illegible] trophy (10 of 30 cases) being associated with manition and general visceral atrophy in most of the cases, or with long standing chronic passive congestion

Chronic passive congestion was frequently present (18 of 30 cases), reflecting a high incidence of serious heart disease.

Leukopenia may exist without splenomegaly and the spleen may be enlarged with a normal leukocyte count.

In many

observed

normal [87] This anemia may be resistant to iron replacement and, in some instances, blood transfusions are necessary to establish a normal level. Recently, it has been demonstrated that *folic acid* improves the anemia of patients with rheumatoid arthritis [102] The rationale of this favorable response is not clearly understood because the bone marrow studies in the author's experience have failed to show any evidence of hyperplasia or increased numbers of macrocytes

Agglutination Tests for Hemolytic Streptococci [19, 42, 69, 77]—Cecil, Nicholls and Stainsby, in 1930,[20] first demonstrated the agglutination reaction for the Streptococcus hemolyticus in the sera of patients with rheumatoid arthritis In a recent article, Cecil and DeGara[19] report that 60 4 per cent of 268 patients with rheumatoid arthritis had a positive *agglutination to the Group A hemolytic Streptococcus* This is a somewhat lower value than first reported in 1930 when 94 per cent of 103 patients

The Streptococcal Agglutination Test in the Blood of Rheumatoid Arthritis[19]

Author	*No Patients Reported*	*Per Cent with Positive Tests*
Ashworth[4]	65	89 2
Blair and Hallman[10]	62	82 0
Cecil and DeGara[19]	268	60 4
Cecil, Nicholls & Stainsby[20]	103	94 0
Clawson, Wetherby, Hilbert and Hilleboe[22]	60	59 5
Cox and *Hill*[23]	41	*71–81*
Dawson, Olmstead & Boots[24]	206	55 0
Dawson, Olmstead & Jost[25]	71	56 3
Ferry and Hunt[34]	36	58 0
Goldie & Griffiths[44]	28	89 0
Gray, Bernhard & Gowen[47]	200	70–76
Gray, Frederick & Gowen[48]	144	69–80
Gray and Gowen[49]	60	75 0
Hartung, Davis, Steinbrocker and Straub[52]	50	36 0
Keefer, Myers and Oppel[66]	22	54 5
McEwen, Bunim and Alexander[65]	36	56 0
Nicholls and Stainsby[73]	110	93 6
Nicholls and Stainsby[76, 77]	613	45 0
Short, Dienes and Bauer[94]	47	53 1
Wainwright[106]	51	54 9
Total	2,903	66 0

Central necrosis was observed in 4 instances, being always associated with rheumatic heart disease and with severe and extremely crippling rheumatoid arthritis Cinchophen was not definitely incriminated in any of the cases.

TABLE 3—Postmortem Observations on the Liver in Thirty Cases of Rheumatoid Arthritis[21]

	Cases
Mean weight (23 cases only) 1,634.6 Gm	
Heaviest (2,765 Gm.)	
Lightest (1,046 Gm.)	
Weighing more than 1,800 Gm	8
Weighing more than 2,000 Gm.	4
Weighing less than 1,600 Gm	10
Weighing less than 1,400 Gm	8
Weighing less than 1,200 Gm.	4
Chronic passive congestion with atrophy of cells about central veins (18 cases)	
Grade 1*	8
Grade 2	9
Grade 3	1
Central necrosis (4 cases)	
Cause of death; rheumatic heart disease	2
Cause of death; pulmonary infections	2
Fatty change (7 cases)	
Grade 1*	2
Grade 2	4
Grade 3	1
Amyloid deposits	1
Subacute yellow atrophy (cinchophen hepatitis)	1
Healed miliary tuberculosis	1
Serous hepatitis (Rossle and Eppinger)	9

*The change was graded on the basis of 1 to 4, in which 1 designates the mildest and 4 the most severe condition

TABLE 4—Postmortem Observations on the Lymph Nodes in Seventeen Cases of Rheumatoid Arthritis[21]

	Cases
Proliferation of reticuloendothelial cells in sinuses	7
Proliferation of reticuloendothelial cells in follicles	1
Suppurative lymphadenitis (2 cases)	
Left inguinal nodes	1
Peripancreatic nodes	1
Degeneration of secondary centers of lymph follicles	2
Amyloid deposits	2

showed positive reactions They believe that the test is "more specific for rheumatoid arthritis than the sedimentation rate of the erythrocytes," and that there was a rough parallelism between the positive agglutination reactions and the sedimentation rate. Dawson[31–35] attached considerable

hat the test was
hands of other
entation rate to

degree[79–82] but in rheumatoid arthritis the effect is demonstrable in considerably higher dilutions. Although sheep erythrocyte agglutination has been studied most extensively, a similar enhancement of agglutination can be shown with erythrocytes of other species[79], with streptococci[107] and presumably with other types of cells. The substance responsible for the effect is present in the globulin fraction of serum. Its heat stability is comparable to that of antibodies but it is not absorbed as readily as antibodies.

This property of rheumatoid arthritis serum was applied as a diagnostic aid by Rose and others.[90] The test which they described compared the titer of the patient's serum for untreated sheep cells with the titer for cells

small to cause
the titers was
or greater were
erous modifica-

tions of this procedure have been described, but that most widely used includes absorption of the patient's serum with unsensitized sheep cells to remove normal agglutinins The titer of the absorbed serum for sensitized cells is then determined. The extent to which absorbed serum agglutinates sensitized cells depends partially upon the degree of sensitization. In the author's experience,[80] when cells were sensitized with one-third of the smallest amount of antisheep cell serum required to cause agglutination, they were rarely agglutinated by the serum of normal individuals to

ent of patients
Patients with
ts with Still's

disease less frequently showed a significant reaction. Titers of 1 in 80 or above have also been obtained in scleroderma, serum disease and severe

Fatty change was found in 7 cases (23 per cent). This lesion was associated with acute infections and with rheumatic heart disease in most instances and with poor nutrition in others.

Serous hepatitis occurred in approximately a third of the cases, usually associated with some degree of passive congestion and atrophy of liver cells. This lesion, a separation of reticulum fibers from the liver cells, has no known significance and may possibly be an artifact resulting from shrinkage of tissue during fixation. (Table 3)

Lymph Nodes.—Lymph nodes do not reveal a lesion pathognomonic of rheumatoid arthritis. Findings in a series of 17 cases are summarized in table 4

This series did not include cases in which striking generalized enlargement of lymph nodes had been present during life. But, moderate hypertrophy in nodes which drained affected joints, was frequently present.

The lesion most commonly responsible for enlargement is hyperplasia of reticuloendothelial cells lying in sinuses and follicles. Suppurative lymphadenitis is found only in nodes draining adjacent infections.

Cellular degeneration in secondary centers of lymph follicles occurs rarely, and amyloid deposits have also been noted in occasional cases. (Table 4)

A notable finding was reported recently in specimens of lymph nodes removed at biopsy [illegible] heumatoid arthritis. This study [illegible] changes which could not be disti[illegible] giant follicular hypertrophy or giant follicular lymphadenopathy In some cases variations of these lesions were observed, distinguished from giant follicular hypertrophy but which seemed to be gradations from this lesion towards the normal This research suggests that this lesion may be an occasional result of the rheumatoid process

The Spleen.—Observations on the spleen are listed in table 5. This organ, like the lymph nodes, reveals no pathognomonic lesion but is frequently diseased Significant enlargement was noted in 58 per cent of cases, [illegible]

[illegible]

centers of some lymph nodes

Amyloid degeneration was found in two cases. (Table 5)

Kidneys.—In nineteen of thirty cases, glomerular endothelial prolifera[illegible]

[illegible]

process However, this was not established with certainty.

[illegible] ving appeared in both

[illegible]

Other renal lesions of passing interest, probably reflecting accidental associations with rheumatoid arthritis, included dissecting aneurysm of the renal artery and nephrolithiasis associated with pyelitis.

Gallbladder.—Lesions found in the gallbladder included chronic cholecystitis in 2 cases (associated with stones in 1 case), uncomplicated chole-

sins) have the same sensitizing capacity even though they may have the

be evaluated in terms of positive and negative controls examined under the same conditions

Antistreptolysin Content of Serum.—Antistreptolysin (antihemolysin or antistreptohemolysin) is an antibody which neutralizes streptolysin; the latter is a soluble product of hemolytic Streptococci which produces hemolysis of erythrocytes. The antistreptolysin titer in the serum is increased in acute hemolytic infections[14] It may be increased in the early stage of

from acute streptococcal infections resist this fibrinolytic action Such resistance is due to the presence of antifibrinolysins The latter are usually present in the blood of patients with acute rheumatic fever, but not with rheumatoid arthritis,[74,78,103] however, when rheumatoid arthritis is well established, the test is negative

Precipitins.—Hemolytic streptococci contain nucleoprotein (P) 38–41 (which is not specific as it gives cross-precipitation with other Gram-positive cocci), a group-specific carbohydrate (C), and a type specific protein and carbohydrate fractions (M and S). The nucleoprotein fractions are precipitated by sera of patients in active rheumatic fever[24] and rheumatoid arthritis.[13,35,82] This test is of little diagnostic value in rheumatoid arthritis

Skin Tests.—Various products and vaccines prepared from hemolytic streptococcus cultures have been employed as skin tests There is no significant data that proves this procedure to be of any value in the diagnosis of rheumatoid arthritis

Mester Test.—This test was said to cause a fall in the leukocyte count thirty minutes after the injection of 0 1 per cent solution of salicylic acid intradermally.[79] A positive reaction consisted of a 15 per cent drop in the white blood count The control group was negative while 98 per cent of the rheumatic group was positive. Lenoch[84] found the test positive in both

arthritis, they considered it grossly unreliable.[80]

Weltmann Coagulation Reaction.—This test was described in 1930 as a means of measuring the activity of rheumatic disease. Kling reported

lithiasis and acute purulent cholecytitis each in 1 case. No relation of these lesions to rheumatoid arthritis was demonstrated. No data were obtained which would be of help in determining whether cholecystitis might act as a focus of infection.

TABLE 5.—Postmortem Observations on the Spleen in Thirty Cases of Rheumatoid Arthritis[n]

		Cases
Mean weight (24 cases)	250 Gm.	
Lightest	86 Gm.	
Heaviest	573 Gm	
Weighing 150-199 Gm.		6
Weighing 200-299 Gm.		5
Weighing 300 Gm and more		9
Chronic passive congestion		8
Reticuloendothelial hyperplasia with atrophy of malpighian corpuscles		5
Amyloid degeneration		2

Gastrointestinal Tract.—Various lesions have been observed in the

acute ulcers in the colon, and mild colitis without ulceration.

Pancreas.—No pancreatic lesions have been encountered which could be related to rheumatoid arthritis. Chronic interstitial pancreatitis, fat necrosis and fatty replacement, have each been found in occasional cases.

Adrenal Glands.—*Adrenal glands have shown no characteristic lesions* ... focal collections of lymphocytes, atrophy, periadrenal arteritis were observed in

Thyroid Gland.—Adenomata have been found in a few patients, and colloid goitre in an equal number. Mild grades of parenchymal hyperplasia have been rarely present

Prostate.—Lesions encountered included suppurative prostatitis and carcinoma. Both lesions were considered coincidental.

characteristic of rheumatoid arthritis has been encountered either in arterial or venous vessels Only moderate grades of arteriosclerosis consistent with ... observed.

...asionally important ...st often in the very ...complication of the ...showing this compli-

results from a series of 250 patients (98 with rheumatoid arthritis), and *found it positive in only 53 per cent,* whereas 82 per cent of the same group had elevated sedimentation rates.[62] Milles and Salt compared the Weltmann reaction, sedimentation rate, formol-gel test and the Takata-Ara test in 150 patients with rheumatoid arthritis.[71] They concluded that the sedimentation rate and the Weltmann reaction were very sensitive indicators of the extent of the activity of rheumatic disease, but that the two did not always parallel each other. In patients with

seemed to them to indicate a favorable prognosis.

As yet, the Weltmann reaction has not proved *itself as a practical laboratory* method. It is, of course, a method indicating the abnormalities in the serum proteins, but these changes are poorly understood and information gained from this test is not sufficient warrant to recommend its use as a routine procedure.

Laboratory Findings in Rheumatoid Arthritis

Sedimentation test—moderately to markedly increased in most cases
Blood count—slight to moderate normocytic normochromic anemia; white blood count normal or slightly decreased in chronic cases—slight to moderate leukocytosis in acute cases, polymorphonuclear leukocytes may be increased with shift to the left
Blood uric acid—normal
Agglutination test—with Group A hemolytic streptococci—positive after several months to a year in the majority of cases; negative in most children and adolescents The strength of the reaction may decrease as improvement occurs
Blood proteins—total proteins—normal, plasma fibrinogen and globulin—increased; albumin—decreased
Blood cholesterol—may be decreased slightly, but usually normal.
Serum calcium and phosphorus—normal
Serum sodium and potassium—normal
Urinary 17 ketosteroids—may be decreased, usually normal
Urinary oxysteroids—usually normal, occasionally may be decreased.
Basal metabolic rate—normal
Blood cultures—negative

Blood Chemistry.—The studies of various chemical components in the blood of patients with rheumatoid arthritis reveal normal values for calcium, phosphorus, phosphatase,[89] uric acid, sugar, creatinin, sulphur,

arthritis and concluded that the calcium and phosphorus metabolism was grossly normal. There was a slight increase in the urinary calcium excretion. Similar control studies were made on 3 patients with degenerative joint disease and they were found to be normal except for a slight decrease



Fig 68
degenerat
Glomerul
× 350
glomerul

in the urinary calcium excretion. This study clearly shows the questionable therapeutic value of agents which affect the calcium metabolism, *i. e.*, parathyroid hormone, vitamin D and A.T 10

Sulfur Metabolism of patients with rheumatoid arthritis is normal.[11, 44]

Protein.—There is a tendency for a reversal of the A. G. ratio and an increased fibrinogen.[3, 10] The fibrinogen and euglobulin content of blood plasma tends to decrease as clinical improvement occurs.[105]

The Glucose Tolerance is normal in over 85 per cent of the patients.[81]

claimed that 50 to 60 per cent of patients with severe rheumatoid arthritis showed liver dysfunction using the Azorubin S, hippuric acid and bilirubin excretion tests [84, 86]

Laboratory Procedures
Routine
1 Red and white blood cell count
2 Differential blood smear.
3 Sedimentation rate of erythrocytes (Westergren, or Wintrobe)
4 Urinalysis (Heat-acetic acid will detect Bence-Jones Protein)
5 Selected roentgen examination of joints (teeth and sinuses only if indicated)
If Indicated
6 Biopsy of subcutaneous nodules, and synovial tissue
7 Serum protein (fasting) (? multiple myeloma).
8 Serum uric acid (fasting) (? gout)
9 Calcium, phosphorus, phosphatase (? hyperparathyroidism, Paget's, malignancy)
10 N P N (blood)—(if patient has been on massive doses of vitamin D).
11 Prostatic smear and culture
12 Blood culture (? bacteremia—subacute bacterial endocarditis)
13 Joint aspiration—with synovial fluid examination
Of Little Value Unless Indicated
14 Basal metabolic rate
15 Electrocardiograph
16. Glucose tolerance test.
17. Bacteriological examination
18 Liver and kidney function tests
19 Congo red test for amyloidosis

Basal Metabolism —There is no abnormality in the basal metabolism

cation had been reported in the world's literature.[21,33] Since that time, occasional cases have been reported from widely separated regions throughout the world.

When amyloid degeneration appears, the blood generally shows hypo-

are severely affected, urea retention usually follows. Most of the cases recognized have died of the amyloid complication, but one recently reported by Trasoff, Schneeberg and Scarf recovered under treatment with liver extract At autopsies severe amyloid involvement of kidneys, liver, spleen, adrenal glands, thyroid and submaxillary glands and lymph nodes has been observed.

Patients affected have ranged in age from early childhood to the eighth decade and males seem somewhat more susceptible than females. Severe loss of weight, wasting and cachexia are generally noted and the patients usually suffer from exhausting bouts of diarrhea.

Hepatomegaly, splenomegaly and edema are the important clinical signs of amyloidosis, but their presence is not essential to the diagnosis in these cases. Among 20 patients with amyloidosis complicating rheumatoid arthritis, 4 showed hepatomegaly, 1
omegaly and in 7 neither spleen nor
may not be present and the blood
albuminuria is an almost invariable feature.

Ocular Lesions.—The eyes are frequently affected in rheumatoid arthritis, and in some instances this complication may bring about partial or even complete blindness The ocular lesions may appear in some cases after articular symptoms are well established, but occasionally the eyes are attacked first; patients with rheumatoid arthritis not infrequently give a history of ocular inflammation which antedated the onset of joint symptoms[16] From the point of view of the pathologist, ocular complications of rheumatoid arthritis strike most frequently in either the uveal tract or in the corneo-scleral layers leaving the retina unaffected. In severe cases, however, the retina may be damaged in consequence of its proximity to lesions which affect the other coats.

The ocular complication most frequently encountered in rheumatoid arthritis is iridocyclitis, or "iritis," an inflammatory lesion primarily involving the corneo-scleral layer. This lesion occurs in at least three to five per cent of all cases Iridocyclitis of rheumatoid arthritis may be mild and transient in some instances or severe, stubborn, and destructive in others Loss of vision from iritis may be negligible in mild cases and severe with total blindness in the intense types.

Less frequently encountered are two varieties of ocular complications which attack primarily the corneo-scleral layer of the ocular bulb. One of these is designated band-shaped opacity of the cornea, and the other, scleromalacia perforans

Band-shaped opacity of the cornea is a rare complication of rheumatoid arthritis.[16,17,19] The condition was first described in 1848[9] as a linear or band-shaped opacity caused by deposition of calcium salts in the inner layers of the cornea. Children and also aged individuals may be affected. When it is found in children, it is almost constantly associated with rheuma-

results from a series of 250 patients (98 with rheumatoid arthritis), and found it positive in only 53 per cent, whereas 82 per cent of the same group had elevated sedimentation rates.[62] Milles and Salt compared the Weltmann reaction, sedimentation rate, formol-gel test and the Takata-Ara test in 150 patients with rheumatoid arthritis.[71] They concluded that the sedimentation rate and the Weltmann reaction were very sensitive indicators of the extent of the activity of rheumatic disease, but that the two did not always parallel each other. In patients with severe rheumatoid arthritis, there was a shift to the left in the serum coagulation band; acute stages of rheumatic fever produce a much greater deviation to the left. An occasional shift to the right in rheumatoid arthritis seemed to them to indicate a favorable prognosis

As yet, the Weltmann reaction has not proved itself as a practical laboratory method. It is, of course, a method indicating the abnormalities in the serum proteins, but these changes are poorly understood and information gained from this test is not sufficient warrant to recommend its use as a routine procedure.

Laboratory Findings in Rheumatoid Arthritis

Sedimentation test—moderately to markedly increased in most cases
Blood count—slight to moderate normocytic normochromic anemia; white blood count normal or slightly decreased in chronic cases—slight to moderate leukocytosis in acute cases, polymorphonuclear leukocytes may be increased with shift to the left.
Blood uric acid—normal.
Agglutination test—with Group A hemolytic streptococci—positive after several months to a year in the majority of cases, negative in most children and adolescents The strength of the reaction may decrease as improvement occurs
Blood proteins—total proteins—normal, plasma fibrinogen and globulin —increased; albumin—decreased.
Blood cholesterol—may be decreased slightly, but usually normal.
Serum calcium and phosphorus—normal
Serum sodium and potassium—normal.
Urinary 17 ketosteroids—may be decreased, usually normal
Urinary oxysteroids—usually normal, occasionally may be decreased
Basal metabolic rate—normal
Blood cultures—negative

Blood Chemistry.—The studies of various chemical components in the blood of patients with rheumatoid arthritis reveal normal values for calcium, phosphorus, phosphatase,[89] uric acid, sugar, creatinin, sulphur, cholesterol, acetylcholine esterase[15] and total protein. The decalcification of the skeleton associated with rheumatoid arthritis has suggested a possible disturbance in the calcium metabolism Ropes, Rossmeisl and Bauer[89] carried out total calcium metabolic studies on 9 patients with rheumatoid arthritis and concluded that the calcium and phosphorus metabolism was grossly normal. There was a slight increase in the urinary calcium excretion. Similar control studies were made on 3 patients with degenerative joint disease and they were found to be normal except for a slight decrease

toid arthritis A similar lesion occasionally found in the advanced years of life is said to follow long-standing iridocyclitis whether from rheumatoid arthritis or from any cause. Pathologic examination in these cases reveals calcific deposits in degenerated, thickened, and eventually fibrosed areas of Bowman's membrane. Chemical studies of this lesion have revealed *quantities of calcium phosphates and carbonates, also sulfur and silicon.*

Pathogenesis of band-shaped opacity is not understood. In one case, the patient had received large doses of Vitamin D for treatment of his

perforans, also designated necroscleritis nodosa and scleritis necroticans This condition was first described by Van der Hoeve in 1930 Scleromalacia perforans is characterized clinically by appearance in the sclera of nodules which later may undergo necrosis and discharge to the outside, leaving perforations through which the uveal coat may bulge. In some instances adjacent perforations coalesce to form larger openings

*Pain is a prominent symptom in some but not in all instances of sclero*malacia perforans These lesions generally appear indolent, being surrounded by little inflammatory reaction

Microscopic appearances of this lesion in its early stages are comparable to subcutaneous nodules from patients with rheumatoid arthritis A central zone of necrosis is seen surrounded by a palisaded wall of epithelioid cells The central zone may be purulent and liquefied [1,12] Beyond the palisaded layer are found accumulations of plasma cells, also fibroblasts. A recent study indicated that the primary lesion is an area of simple necrosis in the sclera which soon becomes surrounded by a wall of epithelioid cells and later pus cells penetrate this wall, entering the interior of the nodule and bringing about the appearance of a central zone of necrosis.

It has been stated that scleromalacia perforans, like iritis, may precede articular symptoms of rheumatoid arthritis

Causes of Death in Rheumatoid Arthritis.—Statistical analysis of the manner in which life terminates among patients with rheumatoid arthritis has not yet been possible, because the disease is not frequently encountered among general-run necropsy material At the Mayo Clinic, for example, several thousand patients with rheumatoid arthritis were studied clinically

This series comprised a group of patients whose ages ranged from 0 to 77 years at time of death Seventeen males and 13 females were included.

myocardial damage, or heart failure resulting from mechanical dysfunction brought about by severe inflammatory deformities of heart valves

he central nervous system showed no specific lesions. Lesions were found n the peripheral nerves in 26 of 31 cases and in the muscles in 8 of 14 cases. These findings were similar to those reported by others.[6,41,43,45,87,101] Similar lesions were found in the peripheral nerves and muscles of 1 case of dermatomyositis; in the nerve of 1 case of disseminated lupus erythematosus and 2 cases of rheumatic fever, and in the muscle from 1 case each of disseminated lupus erythematosus and scleroderma. These non-specific



FIG 60 —A female, aged thirty-eight years, with extensive involvement of all the carpal, metacarpal and many interphalangeal joints There are marked atrophic changes with bony ankylosis involving some of the joints Rheumatoid arthritis

lesions have been demonstrated by others in the muscles in dermatomyositis,[9] scleroderma,[9] rheumatic fever,[11] disseminated lupus erythematosus an [illegible]

[illegible]

to [illegible]

reason, one should not rely too much on this examination in making a diagnosis of rheumatoid arthritis It must be remembered that the primary

d
T

Amyloidosis, an occasional complication of severe rheumatoid arthritis, caused death by inducing widespread anasarca and renal insufficiency.

Pulmonary Deaths.—Chronic pulmonary suppuration was an occasional cause of death but appeared to be an accidental association. Several pulmonary accidents occurring as untoward complications of therapeutic procedures or operations performed for unrelated causes were observed. These included pulmonary embolism, pulmonary fat embolism, bronchopneumonia and postoperative massive collapse of the lungs

Table 6.—Cause of Death in Thirty Cases of Rheumatoid Arthritis[21]

Cause of Death	*Cases*
Cardiac disease (9 cases)	
Rheumatic cardiac disease	7
Non-rheumatic cardiac disease	2
Renal disease (3 cases)	
Acute pyelonephritis with oliguria	2
Renal amyloidosis	1
Pulmonary disease (11 cases)	
Chronic bronchiectasis with pulmonary suppuration	2
Pulmonary embolism	3
Bronchopneumonia	3
Pulmonary fat embolism	2
Postoperative massive collapse	1
Intestinal disease (2 cases)	
Chronic diarrhea of undetermined origin	2
Miscellaneous causes (5 cases)	
Cinchophen hepatitis	1
Violent accidental death	1
Carcinoma of prostate with metastasis	1
Sudden unexplained death	1
Cause of death unknown	1
Total	30

Intestinal Causes of Death.—In 2 instances, death followed severe chronic diarrhea for which no adequate explanation could be found on examination of gastro-intestinal organs at necropsy. It was suspected that the diarrhea had resulted from some functional disturbance of bowel action, possibly caused from a nutritional lack

Miscellaneous Causes of Death.—In this group were placed nearly 17 per cent of the cases Deaths classified as miscellaneous included instances resulting from accidents, cancer, cinchophen hepatitis, and so forth

tissues of patients suffering from rheumatoid arthritis.[3,34,35] Cecil states: *"Carefully controlled studies would seem to indicate that in the majority of cases, the streptococci which have been recovered from the blood and joints in this disease (rheumatoid arthritis) are accidental contaminations.[17]"*

Hormone Excretion Studies.—The excretion of 17-ketosteroids in the urine may be somewhat decreased in rheumatoid arthritis, but is usually normal. Likewise, the urinary oxysteroid excretion is usually low normal or slightly below.

Spinal Fluid Findings.—The spinal fluid is normal in all respects except for an increase in the total proteins and the associated disturbance of the colloidal gold curves. Ludwig, Short and Bauer[66] found the protein to be increased (47 to 105 mg. per cent) in 28.6 per cent of 42 patients with rheumatoid spondylitis (Marie-Strumpell type). This increase was seen in only 6 to 8 per cent of 59 patients with rheumatoid arthritis with peripheral joint involvement. It was concluded that the inflammatory tissue about the facets disturbed the permeability of the meninges and permitted the entrance of protein into the cerebrospinal fluid.

Synovial Fluid in Rheumatoid Arthritis.—Examination of synovial fluid discussed elsewhere shows an exudative reaction with an increase of leukocytes of 600 to 66,000 per cubic millimeters and the preponderance of polymorphonuclear leukocytes (29 to 96 per cent) in a differential smear. Cultural examinations reveal a sterile fluid on aerobic, anaerobic cultures and egg inoculations. The synovial mucin may be denatured in severe disease as demonstrated by its failure to precipitate in a ropy mass with the addition of acetic acid. The fluid is turbulent and resembles the reaction obtained with infected synovial fluids.

Urinalysis.—The examination of the urine by the usual laboratory methods is normal. Patients receiving chrysotherapy and large doses of vitamin D may show evidence of renal disease. The presence of casts and red and white cells in the urinary sediment should suggest the possibility of periarteritis nodosa, acute disseminated lupus erythematosus, subacute bacterial endocarditis and other systemic diseases which involve the arterioles and capillaries. Nephritis is not a manifestation of rheumatoid arthritis with the rare exception of amyloidosis.[2,5,40,53,67,91,96,106]

Subcutaneous Nodules.—The examination of tissue from either the synovial membrane or subcutaneous nodules should always be done to establish the diagnosis in doubtful cases.[8,22,23,26,31,42,54,93,109,110] The microscopic examination of the subcutaneous nodule of rheumatoid arthritis has been shown to have a tendency of the fibroblasts to form a palisade about a central necrotic area. Trained observers can distinguish these nodules from those of rheumatic fever.[8] These authors conclude: *"The nodules of rheumatoid arthritis and rheumatic fever differ as much from one another as do the granulomas of syphilis and tuberculosis, suggesting that they may be due to different agents. The clinical and pathologic differences of rheumatoid arthritis and rheumatic fever also favor this interpretation."*

Non-Specific Inflammatory Lesions in Peripheral Nerves and Muscle Tissue.—Morrison, Short, Ludwig and Schwab[72] have studied the electromyographic and histologic abnormalities of the neuromuscular system in patients with rheumatoid arthritis. The tracings showed no constant deviation from normal controls. Involuntary skeletal activity was inconstantly observed in 50 per cent of 34 patients. Postmortem examination of

pathology is in the synovial membrane where it may remain for long periods of time before the pannus spreads to destroy the joints. Pannus forms at

Bone and articular cartilage destruction appears after months and years have passed and represents the advanced phase of the disease. The first evidence of bone destruction is at the junction of the synovia and the articular cartilage Complete destruction of the articular cartilage is evidenced by a narrowing or absence of the joint space.

In patients with rheumatoid spondylitis, the earliest roentgenographic change occurs in the sacroiliac joints characterized by a haziness of the

tudinal ligaments of the spine.

long-continued effusion causes relaxation of the joint capsule There may also be evidence of soft tissue swelling in the area of the quadriceps pouches above the knee joints and, in some instances, accessory pouches ("Baker's Cysts"), and sometimes thickening about the prepatellar bursæ. The osteoporosis that appears adjacent to the involved joints is demonstrated in Fig 69 As the disease progresses, there may be actual extension of the

tracture. In the advanced disease, there may be complete bony ankylosis of the articulations as demonstrated in the wrist bones of Fig 71.

In some instances, one may see periostitis and calcineal spurs and punched-out areas. Lipping and osteophytes encountered during the examination in individuals after forty years of age should be regarded as degenerative changes consistent with age rather than a manifestation of rheumatoid arthritis It is conceivable that a badly damaged joint would cause the formation of osteophytes, however, this is a secondary development and is not a part of the disease. Punched-out areas of bone destruc-

tion of arthritis and that accurate interpretations cannot be made without the knowledge of the patient's history and other clinical findings (See also chapter on Radiology in Arthritis p. 48)

following reasons:[28] "(1) the totally different pattern of arthritis (one of numerous short attacks and persistent functional restitution); (2) the tendency for only one or two joints to be involved in an attack; (3) the frequent isolated short attacks of para-arthritis, (4) the general absence of significant constitutional reaction, (5) the relative absence of the effect of season and weather, (6) the sedimentation rate which is relatively normal or only moderately and transiently elevated, (7) the moderate increase (rather than decrease) in red blood counts; (8) the persistently negative

separate entity. The author has seen one instance in which severe, crippling rheumatoid arthritis was preceded four years by what appeared to be

... which accompanies acute rheumatic fever is usually intense, of short duration, and completely responds to full, toxic doses of salicylates In rare instances, subjects suffering from rheumatic fever may have symptoms over a period of several months and, in this instance, the possibility of rheumatoid arthritis cannot be completely ruled out. If the sedimentation rate remains elevated and new joints appear after the passage of four to six

... In a study of acute respiratory in-

... patients with non-hemolytic, streptococcus respiratory infections, however, acute rheumatic fever did appear in 15 of the 410 patients who had definite Group A hemolytic streptococcus infections Further study of these 15 cases, prior to the onset of ...
rheumatic fever, by
streptolysins, agglutir
during the so-called "
tory infection and the

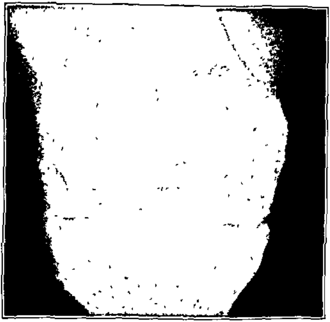


FIG 70 — Rheumatoid arthritis. Marked narrowing of joint space at knee joint indicating destruction of cartilage; homogeneous ground glass appearance in the bones



FIG. 71 — Advanced rheumatoid arthritis Marked osteoporosis, deformities and luxations Destruction of cartilage

articular pain which is not relieved by salicylates and heat, one should suspect the diagnosis of psychogenic rheumatism. A careful history will reveal an inconsistent pattern of the occurrence of the skeletal pain and stiffness. The discomfort may be constant throughout the day, aggravated by emotional disturbances or may disappear during pleasant activities There is frequently a reversal pattern of the arthritic morning pain and stiffness. Even though these patients complain of terrible pain, further questioning reveals that they make no attempt to obtain relief either by using heat or salicylates. Patients suffering from fibrositis or mild rheumatoid arthritis seldom have other complaints except for fatigability and increased nervous tension. When the problem of compensation or insurance settlement clouds the history, *one should not make a diagnosis of* rheumatoid arthritis unless there is undisputed objective evidence of the disease.

Periarticular Fibrositis.—The patient with primary periarticular fibrositis will usually not show the marked loss of weight, anemia, fever, marked muscular atrophy, rapid sedimentation rate and synovial exudate which are features of rheumatoid arthritis.[62] The use of this category should be questioned because it connotes a specific pathological basis for its existence. Thus far there has been no conclusive evidence of a specific histological abnormality that distinguishes fibrositis from other diseases of this group Presumably, there is some physiological abnormality of the connective tissues, muscles or interstitial fluid responsible for rheumatic pain and stiffness. The development of definite subcutaneous nodules is characteristic of rheumatoid arthritis rather than fibrositis. It has been stated that fibrous nodules are characteristic of fibrositis; however, this fact has not been firmly established on the basis of a careful pathological study of these patients. Patients with generalized chronic fibrositis frequently give a history of being under emotional tension for long periods As a rule, these individuals are sensitive, repressed, and pride themselves on not being "nervous," they tend to overload themselves with responsibilities *The terminology "periarticular fibrositis" should be confined to those cases in which there is no associated psychiatric disease, absence of objective laboratory and physical findings consistent with rheumatoid arthritis, and when the symptoms are confined to the musculoskeletal system.* There should also be a distinct pattern of joint symptoms with morning

the patient that he does not have chronic, progressive arthritis.

Palindromic Rheumatism.[28]—Palindromic rheumatism is a rare form of

na of the

ions with

polymorphonuclear leukocytes in large numbers. A fibropurulent exudate

48 GRAY, J. W, FREDERICK, E and GOWEN, C. H: Tex State Jour. Med, 28, 317, 1932
49. GRAY, J. W. and GOWEN, C. H. Am. Jour Med. Sci, 182, 682, 1931
50 GREEN, M. L., and FREYBERG, R. H. Jour Lab & Clin Med, 27, 81, 1941
51 HADEN, R. L. Clinics, 1, 531, 1942
52 HARTUNG, E F, DAVIS, J S, STEINBROCKER, D and STRAUB, M E. Jour Am Med. Assn, 106, 1448, 1936
53 HATCH, F N. Ann [illegible] 72 201 1945
54 HAWTHORNE, C O: [illegible] 1932
55 HENCH, P. S, BAUER [illegible] W, GRAHAM, W., HOLL[illegible] [illegible] BERG, E F and STECHER, R M. Ninth Rheumatism [illegible] 667, 1948
56 HENCH, P S, OSGOOD, R B and WAINWRIGHT, C W Army Med Bull, No 60, 1942
57 HEPBURN, J S, WARTER, P J, and ROSENSTEIN, G Rev Gastroenterol, 10, 126, 1943
58 HILL, L. C Acta Rheumatol., 3, 6, 1931.
59 JORDAN, E P, BAUER, W, BOOTS, R H, CECIL, R L, COGGESHALL, H C, HENCH, P S, KEY, A, LOCKIE, M., MINOT, G R., PEMBERTON, R, and SWAIM, L T Primer on Arthritis, Jour Am Med Assn, 119, 1089, 1942
60 KEEFER, C S, MYERS, W. K and OPPEL, T W. Jour Clin Invest, 12, 267, 1933
61 KERSLEY, G D Proc. Roy Soc Med, 30, 611, 1937
62 KLING, D H Ann. Rheum Dis, 2, 256, 1941
63. LANCEFIELD, R C Proc Soc. Exp Biol and Med, 22, 100, 1924
64 LENOCH, D Wien Klin Wchnschr, 51, 303, 1938
65 LONGCOPE, W T Jour Clin Invest, 15, 277, 1936
66 LUDWIG, A O, SHORT, C L and BAUER, W New England Jour Med, 228, 306, 1943
67 Massachusetts General Hospital Case 31212, New England Jour Med, 232, 632, 1945
68 McEWEN, C, ALEXANDER, R C, and BUNIM, J J Jour Lab & Clin Med, 21, 453, 1936
69 McEWEN, C, BUNIM, J J and ALEXANDER, R. C Jour Lab & Clin Med, 21 465, 1936
70 MESTER, A J Ann Rheum Dis, 2, 192, 1941
71 MILLES, H L, and SALT, H B Ann Rheum Dis, 2, 192, 1941
72 MORRISON, L R., SHORT, C L, LUDWIG, A O and SCHWAB, R S Am. Jour Med Sci, 214, 333, 1947
73 MOTE, J R, and JONES, T D Jour Immunol, 41, 87, 1941
74 MYERS, W K, KEEFER, C S and HOLMES, W F, JR Jour Clin Invest, 14, 119, 1935
75 NICHOLLS, E E and STAINSBY, W J Jour Am Med Assn, 95, 1146, 1931
76 ---- Jour Clin Invest, 10, 323, 1931
77 -- Jour Clin. Invest, 12, 505, 1933
78 OXELL, C C and ELLIOTT, S D Lancet, 2, 869, 1935
79 PIKE, R M, SULKIN, S. E, and COGGESHALL, H C Jour Immunol, 63, 447, 1949
80 -- Jour Immunol, 66, 107, 1950
81 -- To Be Published
82 PIKE, R. M, SULKIN, S E and BURDETTE, R. I To Be Published
83 PITT, G. [illegible] Tr Clin Soc, London, 27, 54, 1893-1894
84 RAWLS, W B Ann Int Med, 12, 1455, 1939
85 RAWLS, W B, GRUSKIN, B J, RESSO, A. H and JORDON, M Jour Lab & Clin Med, 19, 830, 1934
86 RAWLS, W. B, WEISS, S. and COLLINS, V. L Ann Int Med, 10, 1021, 1937
87 REKIN, J H W Jour Am Med Assn., 51, 462, 1908
88 ROBINSON, G. L Ann Rheumat Dis, 3, 207, 1943
89 ROPES, M W, ROSSMEISL, L. C and BAUER, W Jour Clin Invest, 22, 785, 1943

	Rheumatoid Arthritis	Rheumatic Fever	Gonorrheal Arthritis
Age at onset	*90 per cent of cases after age of 15*	*First attack under 15 years in 90 per cent;* may occur at any age	Most often 20 to 40 years, may occur at any age
Preceding upper respiratory infection	Frequent	Present 80-90 per cent	In about 10 per cent
Roentgen-rays	*Characteristic* may be negative early, later generalized osteoporosis, etc.	*Negative except for soft tissue swelling*	May show rapid bony destruction after 14 days
Permanent joint damage	May occur after 6 months	*None*	May be marked
Subcutaneous nodules	Present in about 5 per cent of cases, may persist for weeks	Present in about 15 per cent of cases	Absent
Tenosynovitis	Present in 5 to 10 per cent	Usually absent	Present *in almost 20 per cent of cases*
Response to salicylates	Temporary relief, but no dramatic response	*Usually dramatic* with complete relief of pain and return of temperature to normal *if adequate doses* are used	Not dramatic, some relief of pain and some reduction of temperature
Involvement of pleura or lungs	Rare	Not uncommon	None
Presence of serous conjunctivitis	Absent	Very uncommon	In about 4 per cent of cases
Response to sulfonamides and penicillin	None	None	Excellent within 24 to 72 hours
Complement fixation test (Gc)	Negative	Negative	Positive in over 80 per cent of cases if repeated every 10 days for 3 examinations
Synovial fluid	Sterile	Sterile	Gonococci demonstrable by smear or culture (in at least 25 per cent of cases)
Electrocardiogram	Negative	Evidence of cardiac involvement 25 to 40 per cent	Negative
History of gonorrhea	Usually negative, possibly coincidental	Negative	*Usually within a few weeks preceding joint pains*
Agglutination reaction with hemolytic streptococci	*Strongly positive in most cases of some months' duration*	Usually negative	Negative
Antistreptolysin titer	Normal	Increased	Normal

BIBLIOGRAPHY

1. Aldred-Brown, G. R. P. and Munro, J. M. H.: Quart Jour Med, 4, 269, 1935
2. Andrus, F G.: Minnesota Med, 247, 1071, 1941.
3. Angevine, D. M, Rothbard, Sidney and Cecil, R L: Ann. Rheumat Dis 3, 101, 1942
4. Ashworth, O. O: Virginia Med. Monthly, 59, 452, 1935.
5. Baggenstoss, A. H, and Rosenberg, E. F.: Arch. Path., 37, 54, 1944
6. Bannatyne, G A: Rheumatoid arthritis, Bristol, John Wright, 1898
7. Bauer, W.: New England Jour. Med, 221, 524, 1939.
8. Bennett, G A, Zeller, J W and Bauer, W.: Arch. Path, 30, 70, 1940
9. Black-Shaffer, B Quoted by Steiner, Am Jour. Path, 22, 646, 1946
10. Blair, J E, and Hallman, F A.. J. Clin Invest, 14, 505, 1935
11. Block, W. D and Murrill, W. A: Proc Soc Exp Biol and Med, 47, 374, 1941
12. Boisert, P. L: Jour. Clin Invest, 14, 65, 1940.
13. Bruce, J G, and Caswell, M.. Jour Lab. and Clin. Med, 26, 464, 1934
14. Bunin, J and McEwen, C.: Jour. Clin. Invest, 19, 75, 1940
15. Butt, H. R, Comfort, M. W, Dry, T J. and Osterberg, A. E: Jour Lab and Clin Med., 27, 649, 1942
16. Camp, J D.. Ann Rheumat Dis, 3, 145, 1943.
17. Cecil, R L: Text-Book Med, 7th Ed, Philadelphia, W. B. Saunders Co, 1947.
18. Cecil, R L, Barr, D. P and Du Bois, E. F.: Arch Int Med, 20, 583, 1922
19. Cecil, R L and De Gara, P. F. Am. Jour Med. Sci, 211, 472, 1946
20. Cecil, R. L., Nicholls, E E and Stainsby, W. J. Am Jour Path, 6, 619, 1930
21. Clawson, B J.: Quoted by Steiner, Am. Jour. Path, 22, 646, 1946
22. Clawson, B J. and Wetherby, M.. Am J Path, 8, 283, 1932
23. Clawson, B. J., Wetherby, M., Hilbert, E H, and Hilleboe, H. E: Am Jour. Med Sci, 184, 758, 1932
24. [illegible] Jour Exp Med, 56, 651, 1932.
25. [illegible]
26. [illegible]
27. [illegible]
28. [illegible] Dis, 3, 107, 1942
29. Cox, R [illegible] 1934
30. Davis, J. [illegible]
31. Dawson, [illegible]
32. ——: [illegible] York, Thomas Nelson & Sons, 5, 605, 1935

[illegible] Jour Am Med Assn, 95, 1894, 1930 [illegible] Med, 28, [illegible] 55, 1934 [illegible] 43 [illegible] m Jour [illegible] sn, 113,

44. Freyberg, R. H, Block, W. D. and Fromer, [illegible] 1063, 1939
45. Gibson, H J, Kersley, G D., and Desmarais, M H. L: Ann Rheum Dis, 5, 131, 1946.
46. Goldie, W. and Griffiths, G J Brit Med Jour, 2, 755, 1936
47. Gray, J W, Bernhard, W G, Gowen, C. H.: Am Jour Clin Path, 5, 489, 1935.

the 12 cases studied in detail. In 3 cases, the electrocardiogram was abnormal on the ninth, twenty-third and thirty-sixth, days after the onset of the sore throat and preceded the arthritis by five, nineteen and fifty-two days. These findings emphasize the hypothesis that the clinical manifestation of rheumatic fever may be the result of altered tissue sensitivity to the products of the hemolytic streptococcus. Many observers have contended that the tissue reaction is altered by the initial infection; others believe that repeated infections are necessary for the development of rheumatic fever. Many qualified investigators believe that rheumatic fever is the direct result of a streptococcal infection, but a similar close relationship is less

fifteen years of age. The acute polyarthritis frequently follows a respiratory infection caused by hemolytic streptococcus within one to three weeks. The joint symptoms are of a migratory character and may be so intense that opiates are required to make the patient comfortable. Acute polyarthritis, fever and leukocytosis subside dramatically within 24 to 72 hours after administration of full, toxic doses of salicylates. The appearance of myocarditis, pericarditis, pleuritis and chorea are characteristic of rheumatic fever. The antistreptolysin and antifibrolysin titres are usually increased in rheumatic fever and normal in rheumatoid arthritis. The various serological reactions and agglutination reactions to hemolytic streptococcus are not practical for the average practitioner and are not considered as necessary criteria to differentiate these two diseases.

There are similarities between rheumatoid arthritis and rheumatic fever ... To date, there is not ... are ... it of

ture *The Aschoff's nodules, characteristic of rheumatic fever, have never been demonstrated in the cardiac muscle of subjects dying from juvenile rheumatoid arthritis* (Still's disease). Valvular lesions similar to those of rheumatic fever have been found in the postmortem examinations of patients with rheumatoid arthritis which suggests that these diseases are: (1) the same, (2) concurrent or, (3) that rheumatoid arthritis is capable of producing valvular disease. *Until the etiology of these two diseases is better understood, it is better to consider these two conditions as separate entities which may be occasionally found in the same individual.*

Postscarlatinal Arthritis.—A mild polyarthritis may appear during the convalescent period of scarlet fever. There may be a low-grade fever, debility and a slightly elevated sedimentation rate. Patients may have a purulent arthritis with hemolytic Streptococci cultured from the joint fluid, acute rheumatic fever, or a mild arthralgia which may persist for months.

Gonorrheal Arthritis.[4, 5, 12]—Gonorrheal arthritis is a true focal infectious arthritis which is a metastatic complication of the same infection elsewhere in the body. A careful history will reveal that the acute arthritis appears within one to three weeks after the initial genito-urinary infection. The onset is characterized by a bacteremia with a chilly feeling, fever and joint

CHAPTER 14

RHEUMATOID ARTHRITIS: DIFFERENTIAL DIAGNOSIS

By Howard C. Coggeshall, M.D.

The typical case of rheumatoid arthritis with a symmetrical, progressive joint disease offers little difficulty in diagnosis. In the atypical and the early stages of the disease, one may encounter difficulty and, in some instances, one is forced to "wait and see" further developments.

Atypical Rheumatoid Arthritis.—This group includes a large number of patients who have atypical manifestations of chronic rheumatoid arthritis. In many instances one may see a mild arthritis, asymmetrical joint involvement and mild skeletal symptoms without objective joint findings. An acute, explosive onset of bursitis and tendinitis may precede the development of a mild, typical arthritis. Frequently it is difficult to differentiate [illegible]

are listed below:

1. Psychogenic rheumatism (psychoneurosis manifested by skeletal pain)
2. Periarticular fibrositis, chronic.
3. Palindromic rheumatism or palindromic phase of rheumatoid arthritis
4. Subacute or recurrent rheumatic fever
5. Postscarlatinal arthralgia
6. Gonorrheal arthritis
7. Reiter's syndrome
8. Gouty arthritis
9. Degenerative joint disease (osteoarthritis)
10. Tuberculous arthritis.
11. Focal infection
12. Syphilitic joint disease (Clutton's Joints)
13. Traumatic arthritis.
14. Lymphogranuloma venereum
15. Acute disseminated lupus erythematosus.
16. Periarteritis nodosa

Psychogenic Rheumatism.—[5,9,15,22,23,26,27,40] One of the most disturbing problems to the general practitioner is the large group of patients who complain of skeletal pains which accompany symptoms associated with psychoneurosis and neurocirculatory asthenia. Some authorities[45] object to the use of the term "psychogenic rheumatism" because they believe that the skeletal symptoms are merely a manifestation of a psychiatric disturbance such as hypochondriasis, hysteria and psychoneurosis. It is the feeling of the author that this diagnosis is just as acceptable as the term "cardiac neurosis" and "gastric neurosis." Many of these individuals complain of bizarre symptoms which fail to fit into the expected pattern of either primary fibrositis or early rheumatoid arthritis. Approximately 15 to 42 per cent of soldiers admitted to various military hospitals complaining of rheumatic pains have psychogenic rheumatism.[5,9,15,23,26,27,40]

Psychogenic rheumatism must be differentiated from primary fibrositis and mild rheumatoid arthritis. Whenever a patient complains of constant

1. *Gout occurs usually in males* (98 per cent of the cases).
2. Gout is *uncommon under* the age of *thirty-five*
3. [illegible]

between attacks.

4. The complete freedom from joint symptoms between attacks of gout is characteristic of this disease. Attacks may occur every year or two at first, and later increase in frequency, so that they may finally come on every week, month or more often
5. The *dramatic response* of the patient to adequate doses of *colchicine* is a [illegible]

such therapy, suspect that the diagnosis is incorrect (unless the dose has been inadequate or the medication has not been retained)

6. The first attack of gout is usually *monarticular*, frequently with involvement of a great toe.
7. *Involvement of* the *great toe* occurs in 70 per cent of cases in the initial attack and sooner or later in about 90 per cent of patients
8. *Tophi* may be seen in 50 per cent of cases
9. The *serum uric acid* is usually elevated above 6 mg. especially during attacks of the disease
10. Generalized osteoporosis, seen by x-rays in rheumatoid arthritis, is not [illegible]
11. [illegible]
12. [illegible] swelling is irregular and asymmetrical.
13. The subcutaneous nodules often seen with rheumatoid arthritis are not found in gout, although the olecranon bursa may be filled with uric acid crystals
14. The patient is usually overweight in contradistinction to the usual patient with rheumatoid arthritis who is weak, tired, underweight, anemic and asthenic.
15. Ankylosis is very rare in gout, while this does occur in the later stages of rheumatoid arthritis
16. In gout, joint swelling, redness and pain may develop very suddenly, in a period of minutes or hours, while the typical joint changes in rheumatoid arthritis consume weeks and months
17. Muscular atrophy, which is so common in rheumatoid arthritis, is not a feature of gout.
18. Most patients with gout give a family history of the disease.
19. The pain in gouty patients is often excruciating and much greater than the discomfort seen in most individuals with rheumatoid arthritis.

Degenerative Joint Disease.—Although it is usually relatively easy to [illegible]

the symptoms of acute generalized progressive rheumatoid arthritis for the first time in their lives. In such cases, the diagnosis is made on the basis of

90. Rose, H. M., Ragan, C., Pearce, E., and Lipman, M. O: Proc. Soc Exp. Biol and Med, 68, 1, 1948.
91. Rosenberg, E F, Baggenstoss, A H. and Hench, P. S.: Ann Int Med., 20, 903, 1944
92. Scott, S G: Radiological Atlas of Chronic Rheumatic Arthritis, Oxford Med Pub, London, Oxford Univ Press, 1935
93. Seegel, D. H., Heidelberger, M., Jost, E L., Little, J. D · Proc Soc Exp Biol and Med, 30, 582, 1933
94. Short, C. L.· Med Bull N Africa Theat. Op, 1, 10, 1944
95. Short, C L, Dienes, L and Bauer, W.: Jour. Am Med Assn, 108, 2087, 1937
96. Solomon, W M. Ann Int. Med, 18, 846, 1943
97. Sparks, M I and Haden, R L: Am. Jour. Med Sci, 184, 753, 1932
98. Steinberg, C. L. Jour Missouri M A., 30, 485, 1933.
99. ——— Am Jour Med Sci, 190, 98, 1935
100. Steinbrocher, O, Hartung, E. F: Jour. Am. Med. Assn., 100, 654, 1933
101. Steiner, G · Am Jour. Path, 22, 646, 1946
102. Stephens, A L, Jr, Borden, W, Holbrook, W. P. and Hill, D F: Ann. Int Med, 27, 420, 1947
103. Stuart-Harris, C H: Lancet, 2, 1456, 1935 Brit Jour. Exp Path, 16, 513, 1935
104. Swartz, M. and Schlossmann, K Acta Med Scand, 142, 420, 1952.
105. [illegible] 19, 426, 1944
106. [illegible] Med, 74, 1944
107. [illegible]
108. [illegible], 1934 Bull Johns Hopkins Hosp, 61, 358, 1937
109. Weber, F P: Ann. Rheumat Dis, 4, 3, 1944.
110. ——— Brit Jour. Dermat, 55, 1, 1943

pain. In the beginning, there is a polyarthritis but later it tends to be a monoarthritis. In only approximately 25 per cent of the cases will the infection be severe enough to cause an infected synovial fluid. Examination of an infected synovial fluid will show a low or absence of sugar (fasting), leukocyte counts in excess of 40,000 per cubic millimeters, and a predominance of polymorphonuclear leukocytes. The presence of a positive gonococcal complement fixation test of the serum and synovial fluid is suggestive, but not diagnostic. The prompt administration of chemotherapy in the form of penicillin, sulfonamides, aureomycin, chloromycetin, and other antibiotic agents is followed by a prompt relief of symptoms These drugs will sterilize the synovial fluid within twenty-four to seventy-two hours. If there is not a prompt response, the type of chemotherapy should be changed to one of the other forms in order to rule out the possibility that the gonococcus might be resistant to the particular form applied

Gonorrheal arthritis does not progress into a chronic form resembling rheumatoid arthritis. When such an instance occurs, it should be suspected that the genito-urinary infection precipitated rheumatoid arthritis. One should rule out gonorrheal arthritis if there is new joint involvement following adequate chemotherapy.

Diagnostic Pointers in Gonorrheal Arthritis

- *History of gonorrhea* (urethritis or involvement of the cervix, conjunctiva or rectum).
- Onset of joint symptoms *usually* within ten to twenty days after primary gonorrheal infection.
- Predominance in males (3:1)
- Usually a *polyarthritis at the onset*
- Involvement of *knees, ankles* and *wrists* in order of frequency.
- Frequent occurrence of tenosynovitis about the wrists and ankles.
- Occasionally associated with conjunctivitis and iridocyclitis.
- *Gonococcus complement fixation test of blood* positive in over 80 per cent of cases (if properly performed); may remain negative for the first four weeks of the disease
- Isolation of gonococci from joint fluid (by special culture methods)
- Prompt response to penicillin and sulfonamides
- Failure of complete response (arthritis and fever) to salicylates

Reiter's Syndrome.[3]—Reiter's syndrome is characterized by acute urethritis, conjunctivitis and arthritis, runs a self-limited course, and is not related to a gonococcal infection. Occasionally in rheumatoid arthritis, one [illegible]

point in the differential diagnosis. (See also Chapter 44.)

Gouty Arthritis.—Gout is occasionally confused with rheumatoid arthritis or gonorrheal arthritis. A few pointers will usually suffice in making the correct diagnosis.

CHAPTER 14

RHEUMATOID ARTHRITIS: DIFFERENTIAL DIAGNOSIS

By Howard C. Coggeshall, M.D.

stances, one is forced to "wait and see" further developments.

Atypical Rheumatoid Arthritis.—This group includes a large number of patients who have atypical manifestations of chronic rheumatoid arthritis. In many instances one may see a mild arthritis, asymmetrical joint involvement and mild skeletal symptoms without objective joint findings. An acute, explosive onset of bursitis and tendinitis may precede the develop-

Some of the types of arthritis which may resemble rheumatoid arthritis are listed below:

1. Psychogenic rheumatism (psychoneurosis manifested by skeletal pain)
2. Periarticular fibrositis, chronic.
3. Palindromic rheumatism or palindromic phase of rheumatoid arthritis
4. Subacute or recurrent rheumatic fever
5. Postscarlatinal arthralgia
6. Gonorrheal arthritis
7. Reiter's syndrome
8. Gouty arthritis
9. Degenerative joint disease (osteoarthritis).
10. Tuberculous arthritis
11. Focal infection
12. Syphilitic joint disease (Clutton's Joints)
13. Traumatic arthritis
14. Lymphogranuloma venereum
15. Acute disseminated lupus erythematosus.
16. Periarteritis nodosa

Psychogenic Rheumatism.—[8,9,18,22,23,26,27,40] One of the most disturbing problems to the general practitioner is the large group of patients who complain of skeletal pains which accompany symptoms associated with psychoneurosis and neurocirculatory asthenia. Some authorities[15] object to the use of the term "psychogenic rheumatism" because they believe that the skeletal symptoms are merely a manifestation of a psychiatric disturbance

bizarre symptoms which fail to fit into the expected pattern of either primary fibrositis or early rheumatoid arthritis. Approximately 15 to 42 per cent of soldiers admitted to various military hospitals complaining of rheumatic pains have psychogenic rheumatism.[8,9,18,23,26,27,40]

Psychogenic rheumatism must be differentiated from primary fibrositis and mild rheumatoid arthritis. Whenever a patient complains of constant

1. *Gout occurs usually in males* (98 per cent of the cases).
2. Gout is *uncommon under* the age of *thirty-five*.
3. In typical cases, the attacks of gout appear suddenly in a previously healthy individual, persist for a few days or weeks, and then disappear, leaving the patient without any joint symptoms during the periods between attacks.
4. **The complete freedom from joint symptoms between attacks of gout is characteristic of this disease.** Attacks may occur every year or two at first, and later increase in frequency, so that they may finally come on every week, month or more often.
5. The *dramatic response* of the patient to adequate doses of *colchicine* is a [illegible]

6. The first attack of gout is usually *monarticular*, frequently with involvement of a great toe.
7. *Involvement of* the *great toe* occurs in 70 per cent of cases in the initial attack and sooner or later in about 90 per cent of patients.
8. *Tophi* may be seen in 50 per cent of cases
9. The *serum uric acid* is usually elevated above 6 mg especially during attacks of the disease.
10. Generalized osteoporosis, seen by *x*-rays in rheumatoid arthritis, is not a feature of gout
11. As differentiated from rheumatoid arthritis, *joint involvement* is usually *not symmetrical* in gout.
12. Fusiform swellings of joints do not usually appear in gout, but the joint swelling is irregular and asymmetrical
13. The subcutaneous nodules often seen with rheumatoid arthritis are not found in gout, although the olecranon bursa may be filled with uric acid crystals
14. The patient is usually overweight in contradistinction to the usual patient with rheumatoid arthritis who is weak, tired, underweight, anemic and asthenic.
15. Ankylosis is very rare in gout, while this does occur in the later stages of rheumatoid arthritis
16. In gout, joint swelling, redness and pain may develop very suddenly, in a period of minutes or hours, while the typical joint changes in rheumatoid arthritis consume weeks and months.
17. Muscular atrophy, which is so common in rheumatoid arthritis, is not a feature of gout.
18. Most patients with gout give a family history of the disease.
19. The pain in gouty patients is often excruciating and much greater than the discomfort seen in most individuals with rheumatoid arthritis.

Degenerative Joint Disease.—Although it is usually relatively easy to [illegible]

the symptoms of acute generalized progressive rheumatoid arthritis for the first time in their lives. In such cases, the diagnosis is made on the basis of

articular pain which is not relieved by salicylates and heat, one should suspect the diagnosis of psychogenic rheumatism. A careful history will reveal an inconsistent pattern of the occurrence of the skeletal pain and stiffness. The discomfort may be constant throughout the day, aggravated by emotional disturbances or may disappear during pleasant activities. There is frequently a reversal pattern of the arthritic morning pain and stiffness. Even though these patients complain of terrible pain, further questioning reveals that they make no attempt to obtain relief either by using heat or salicylates. Patients suffering from fibrositis or mild rheumatoid arthritis seldom have other complaints except for fatigability and increased nervous tension When the problem of compensation or insurance settlement clouds the history, one should not make a diagnosis of rheumatoid arthritis unless there is undisputed objective evidence of the disease.

Periarticular Fibrositis.—The patient with primary periarticular fibrositis will usually not show the marked loss of weight, anemia, fever, marked muscular atrophy, rapid sedimentation rate and synovial exudate which are features of rheumatoid arthritis.[52] The use of this category should be questioned because it connotes a specific pathological basis for its existence. Thus far there has been no conclusive evidence of a specific histological abnormality that distinguishes fibrositis from other diseases of this group Presumably, there is some physiological abnormality of the connective tissues, muscles or interstitial fluid responsible for rheumatic pain and stiffness. The development of definite subcutaneous nodules is characteristic of rheumatoid arthritis rather than fibrositis. It has been stated that fibrous nodules are characteristic of fibrositis; however, this fact has not been firmly established on the basis of a careful pathological study of these patients. Patients with generalized chronic fibrositis frequently give a history of being under emotional tension for long periods As a rule, these individuals are sensitive, repressed, and pride themselves on not being "nervous," they tend to overload themselves with responsibilities. *The terminology "periarticular fibrositis" should be confined to those cases in which there is no associated psychiatric disease, absence of objective laboratory and physical findings consistent with rheumatoid arthritis, and when the symptoms are confined to the musculoskeletal system* There should also be a distinct pattern of joint symptoms with morning [illegible] and relief with application of heat

[illegible] iagnosis

[illegible] ıssuring

the patient that he does not have chronic, progressive [illegible].

Palindromic Rheumatism.[28]—Palindromic rheumatism is a rare form of recurring afebrile attacks of acute arthritis. The word "palindromic" was derived from the Greek word meaning "recurring" or "running-back to."

[illegible]

and last only a few hours or days with complete recovery. [illegible] rheumatism may simulate gouty arthritis.

[illegible] edema of the

	Rheumatoid Arthritis	Gout
Sex	*2 or 3 females to each male*	*98 per cent of cases are males*
Onset	Usually insidious	Attacks usually appear suddenly
Age of onset	Usually between 20 to 45 years	Usually after 35 years
Frequency of attacks	Onset usually insidious, recurrent attacks occasionally early in the disease	Varying from several months to several years
Freedom from symptoms	Symptoms usually continue for months or years without marked intermissions	Usually complete freedom from symptoms between attacks
Relief of symptoms by full dose of colchicine	None	Usually prompt
Blood uric acid	Usually normal	Usually elevated during attacks
Tophi	None	Present in 50 per cent of cases
Involvement of big toe	Not characteristic	Occurs in 70 per cent in initial attack and sooner or later in 90 per cent
Generalized osteoporosis by *x*-ray	Usually seen	Not a feature
Symmetrical joint swellings	Usually present	Not usual
Fusiform joint swelling	Usually present	Joint swelling usually asymmetrical and irregular
Sudden development of joint symptoms in *minutes or hours*	Does not occur this suddenly	Common
Associated olecranon bursitis	Not common	Common
Subcutaneous nodules	In 5 per cent	Not found
Prodromes	Often marked (fatigue, paresthesias, cold, clammy hands)	Usually none
Type of patient	Usually thin, asthenic	Often overweight sthenic
Muscular atrophy	Usually present and may be marked	Usually none except terminal stages
Ankylosis	May occur	Rare
Agglutination test with hemolytic streptococci	Often positive after some months of illness	Negative
Family history of gout	Usually negative	Often positive
No of joints involved	Usually multiple	First attack is usually monarticular
Pain	Usually mild, relieved by rest	Often excruciating, relieved by adequate doses of colchicine

may appear within the cavity of the joint

following reasons.[23] "(1) the totally different pattern of arthritis (one of numerous short attacks and persistent functional restitution); (2) the tendency for only one or two joints to be involved in an attack; (3) the frequent isolated short attacks of para-arthritis; (4) the general absence of significant constitutional reaction;
season and
or only mo
(rather than decrease) in red blood counts; (8) the persistently negative

was preceded four years by what appeared to be

completely ruled out. If the sedimentation rate remains elevated and new joints appear after the passage of four to six months, one should question the diagnosis
unusual th
rheumatic
failed to ol
The prima
response to

Group A hemolytic streptococcus infections
Further study of these 15 cases, prior to the onset of the
rheumatic fever, by
streptolysins, agglutir
during the so-called "
tory infection and the

	Rheumatoid Arthritis vs.	Degenerative Joint Disease
Synonyms	Atrophic, chronic infectious; proliferative	Hypertrophic; osteo-arthritis, degenerative arthritis
Age of onset	[illegible]	[illegible]
Prodromes	[illegible]	[illegible]
Involvement of hands	[illegible] joints	[illegible] erden's nodes)
Other joints commonly involved	Generalized	Knees, lumbar spine, sacro-iliac, lower cervical spine, hips
No of joints involved	Usually many	Usually few
Symmetry of joint involvement	Most often symmetrical	Often asymmetrical
Type of patient	Usually thin, asthenic, visceroptotic	Often overweight, sthenic
[illegible]	[illegible]	[illegible] ular or knobby
Muscular atrophy	Usually present and may be marked	Not frequent, not marked
Skin changes	Cold, clammy hands, smooth, shiny skin in extremities	None except those of age
Joint effusion	Usually present, may be marked	Usually none
Subcutaneous nodules	Present in 5 per cent	Absent
Sedimentation rate	Usually rapid	Usually normal
X-ray findings	Early fusiform periarticular soft tissue swelling, rarefaction of trabeculated ends of bones and systemic decalcification Later decrease of joint space, lip- [illegible]	Early narrowing of the joint space due to destruction of cartilage, formation of osteophytes and *lipping* Later cysts at ends of [illegible] es from [illegible] raction, [illegible]
Ankylosis	[illegible]	[illegible]
Primary pathological change	Synovitis	Degeneration of hyaline articular cartilage, no inflammatory changes
Agglut test with hemolytic streptococci	Strongly positive in over 50 per cent of cases after several months of illness	Usually negative
Progress of the disease	Marked tendency to progression despite treatment	Slow progress, often arrested by supportive measures
Prognosis	Very doubtful, 10 per cent progress to ankylosis and invalidism despite good therapy	Usually good as regards function, usually no crippling, *no flexion deformities*

articular pain which is not relieved by salicylates and heat, one should suspect the diagnosis of psychogenic rheumatism. A careful history will reveal an inconsistent pattern of the occurrence of the skeletal pain and stiffness The discomfort may be constant throughout the day, aggravated by emotional disturbances or may disappear during pleasant activities There is frequently a reversal pattern of the arthritic morning pain and stiffness. Even though these patients complain of terrible pain, further questioning reveals that they make no attempt to obtain relief either by using heat or salicylates Patients suffering from fibrositis or mild rheumatoid arthritis seldom have other complaints except for fatigability and increased nervous tension. When the problem of compensation or insurance settlement clouds the history, one should not make a diagnosis of rheumatoid arthritis unless there is undisputed objective evidence of the disease

Periarticular Fibrositis.—The patient with primary periarticular fibrositis will usually not show the marked loss of weight, anemia, fever, marked muscular atrophy, rapid sedimentation rate and synovial exudate which are features of rheumatoid arthritis.[32] The use of this category should be questioned because it connotes a specific pathological basis for its existence Thus far there has been no conclusive evidence of a specific histological abnormality that distinguishes fibrositis from other diseases of this group Presumably, there is some physiological abnormality of the connective tissues, muscles or interstitial fluid responsible for rheumatic pain and stiffness The development of definite subcutaneous nodules is characteristic of rheumatoid arthritis rather than fibrositis. It has been stated that fibrous nodules are characteristic of fibrositis; however, this fact has not been firmly established on the basis of a careful pathological study of these patients Patients with generalized chronic fibrositis frequently give a history of being under emotional tension for long periods. As a rule, these individuals are sensitive, repressed, and pride themselves on not being "nervous," they tend to overload themselves with responsibilities. *The terminology "periarticular fibrositis" should be confined to those cases in which there is no associated psychiatric disease, absence of objective laboratory and physical findings consistent with rheumatoid arthritis, and when the symptoms are confined to the musculoskeletal system.* There should also be a distinct pattern of joint symptoms with morning stiffness, limbering up following exercise, and relief with application of heat and salicylate medication. It should also be appreciated that this diagnosis is not firmly established but is one of convenience and is useful in reassuring the patient that he does not have chronic, progressive arthritis.

Palindromic Rheumatism.[33]—Palindromic rheumatism is a rare form of

arthritis and periarthritis with pain, swelling and redness of generally only one small or large joint. The attack may appear suddenly, develop rapidly, and last only a few hours or days with complete recovery. Palindromic rheumatism may simulate gouty arthritis.

Examination of synovial tissues dur[illegible] edema of the periarticular tissues, thickening of the polymorphonuclear leukocytes in large

	Rheumatoid Arthritis	Gout
Sex	2 or 3 females to each male	98 per cent of cases are males
Onset	Usually insidious	Attacks usually appear suddenly
Age of onset	Usually between 20 to 45 years	Usually after 35 years
Frequency of attacks	Onset usually insidious, recurrent attacks occasionally early in the disease	Varying from several months to several years
Freedom from symptoms	Symptoms usually continue for months or years without marked intermissions	Usually complete freedom from symptoms between attacks
Relief of symptoms by full dose of colchicine	None	Usually prompt
Blood uric acid	Usually normal	Usually elevated during attacks
Tophi	None	Present in 50 per cent of cases
Involvement of big toe	Not characteristic	Occurs in 70 per cent in initial attack and sooner or later in 90 per cent
Generalized osteoporosis by *x*-ray	Usually seen	Not a feature
Symmetrical joint swellings	Usually present	Not usual
Fusiform joint swelling	Usually present	Joint swelling usually asymmetrical and irregular
Sudden development of joint symptoms in minutes or hours	Does not occur this suddenly	Common
Associated olecranon bursitis	Not common	Common
Subcutaneous nodules	In 5 per cent	Not found
Prodromes	Often marked (fatigue, paresthesias, cold, clammy hands)	Usually none
Type of patient	Usually thin, asthenic	Often overweight, sthenic
Muscular atrophy	Usually present and may be marked	Usually none except terminal stages
Ankylosis	May occur	Rare
Agglutination test with hemolytic streptococci	Often positive after some months of illness	Negative
Family history of gout	Usually negative	Often positive
No of joints involved	Usually multiple	First attack is usually monarticular
Pain	Usually mild, relieved by rest	Often excruciating, relieved by adequate doses of colchicine

[illegible]

	Rheumatoid Arthritis vs.	Degenerative Joint Disease
Synonyms	Atrophic, chronic infectious, proliferative	Hypertrophic, osteo-arthritis, degenerative arthritis
Age of onset	Usually between 20 to 40 years	Most often after 40 years
Prodromes	Often marked fatigue, nervousness, chilliness, numbness and tingling in extremities, etc	Usually none
Involvement of hands	Especially proximal interphalangeal and metacarpophalangeal joints	Especially terminal interphalangeal joints (Heberden's nodes)
Other joints commonly involved	Generalized	Knees, lumbar spine, sacro-iliac lower cervical spine, hips
No of joints involved	Usually many	Usually few
Symmetry of joint involvement	Most often symmetrical	Often asymmetrical
Type of patient	Usually thin, asthenic, visceroptotic	Often overweight, sthenic
Type of onset	Usually insidious, may be abrupt	Usually insidious
Constitutional symptoms	Fever, loss of weight, anemia tachycardia, often enlargement of lymph nodes and spleen	Usually none
Type of joint swelling	Usually fusiform or spindle shaped	Usually irregular or knobby
Muscular atrophy	Usually present and may be marked	Not frequent, not marked
Skin changes	Cold, clammy hands, smooth shiny skin in extremities	None except those of age
Joint effusion	Usually present, may be marked	Usually none
Subcutaneous nodules	Present in 5 per cent	Absent
Sedimentation rate	[illegible]	[illegible]
X-ray findings	[illegible] systemic decalcification Later decrease of joint space, lipping and osteophytes, punched-out areas of bone at articular margins and possibly ankylosis subluxations etc	[illegible] the [illegible] de- [illegible] lage formation of osteophytes and lipping Later cysts at ends of bone, deformities from pressure and traction, no ankylosis
Ankylosis	In about 25 per cent in later stages	No true ankylosis
Primary pathological change	Synovitis	Degeneration of hyaline articular cartilage, no inflammatory changes
Agglut test with hemolytic streptococci	[illegible]	[illegible]
Progress of the disease	[illegible]	[illegible] often ar- [illegible] supportive measures
Prognosis	Very doubtful 10 per cent progress to ankylosis and invalidism despite good therapy	Usually good as regards function, usually no crippling, no flexion deformities

pleura should be suspected when pulmonary osteoarthropathy is associated with articular symptoms

Usual findings are: clubbed fingers and/or toes and other soft tissue changes associated with subperiosteal symmetrical proliferation of bone, usually affecting the bones of the arms or legs, but also observed in the clavicle, ribs and pelvis.[48]

In some cases, sclerosis and thickening of the cortex of phalanges are noted on *x*-ray.

Curving of the nails (parrot beaking) is usually an early sign

Atrophy of the cancellous tufts of phalanges with elongation of the

involvement, synovitis, erosion of articular surfaces or ankylosis

No single hypothesis adequately explains the syndrome

	Rheumatoid arthritis	Buerger's disease	Scleroderma	Raynaud's disease
Age of onset	20 50 most often	20 50	20 55	20 45
Sex incidence	2 or 3 females to 1 male	95% *males*	Equal	95% *females*
Pulses	Present	1 or more —*absent*	Present	Present
Vascular tone	High	Low or high	High	High
Color changes	Variable	Pallor or cyanosis	May have blanching	White then cyanotic then red *Bilateral symmetrical*
Roentgen findings	*Typical* narrowed joint space demineralization of bones fusiform joint swelling etc	Negative	May have *calcification in periarticular soft tissues and absorption of terminal phalanges* in advanced cases	Negative
Sedimentation rate	*Rapid*	Normal	Normal	Normal
Clinical response to heat	Improved	No change	Questionable	Improved
Dissociated vascular response in digits	*Present*	No dissociation	No dissociation	No dissociation
Joint changes—clinically	*Fusiform joint swellings*	None	May have atrophic changes and periarticular soft tissue calcification	Occasional subjective sense of swelling, usually no objective swelling
Pigmentation of skin	May occur late	None	May occur	None
Pain	*Present*	Present	*Usually none* unless ulcers form	Present
Tight leathery skin	Skin may be taut	Absent	*Present*	Absent
Loss of hair in involved extremities	None	None	*Usually marked*	None

Leukemia:

This is often mistaken for rheumatic fever in young children, diagnosis is made by the finding of an abnormal leukocyte pattern; may require a sternal marrow biopsy.

Polycythemia Vera:

Joint pains and limitation of motion may occur [14]

Gout occasionally appears in these individuals Serum uric acid is often elevated (secondary gout)

Neuralgic pains in the extremities and muscular spasms may occur.

This is diagnosed by the marked increase in erythrocytes and hemoglobin, red cyanosis, enlarged spleen, etc.

Roentgen Differentiation of Rheumatoid Arthritis from Gout

Punched out areas may occur in both rheumatoid arthritis and gout.

When surrounded by a sharply circumscribed swelling in the soft [illegible] of cartilage, and a more asymmetrical distribution of joint swelling.

Bony cysts are usually larger in gout than in rheumatoid arthritis.

Gout is often localized in the metatarsophalangeal joints of the big toe, finger joints and wrist.

Roentgen Differentiation of Rheumatoid Arthritis and Degenerative Joint Disease (Osteo-arthritis)

	Rheumatoid Arthritis	Degenerative Joint Disease
Earliest signs	Fusiform swelling of soft tissues and rarefaction of trabeculated ends of bones	Formation of osteophytes and narrowing of the joint space
Soft tissue involvement	Fusiform swelling about joints, especially in phalanges	Usually none
Lipping and osteophytes	In about 75 per cent of cases	In every case
Heberden's nodes	None	Very frequent
Punched out areas of bone at articular margins	Very common	Usually absent
Systemic decalcification	In almost every case; may be the earliest roentgen sign	Mild and consistent with age of patient
Narrowing of joint space	In almost every case	In about 50 per cent of cases
Ankylosis (fibrous and bony)	In approximately 25 per cent of cases	Very rare; only in spine as a locking of osteophytes

presence of fever, anemia, generalized lymphadenopathy, splenomegalia, muscular atrophy, joint effusion, subcutaneous nodules, ankylosis and marked increase in sedimentation rate. In such an individual who presents [illegible] of osteophytes and [illegible] primarily rheumatoid arthritis, the term "mixed arthritis" should not be be used because it fails to give a clear-cut diagnosis of the existing condition. One should not be misled in making the diagnosis of degenerative joint disease on the basis of osteophytes and other roentgenographic evidence of degenerative disease which occurs with increasing regularity in each decade after the age of forty.

A distinction between these two diseases can almost always be made on the basis of the history, alone. Pathologically, they are distinctly different

Erythralgia (formerly called erythromelalgia):

This is characterized by burning pain in the extremities (especially the [illegible]

Raynaud's Syndrome:

This is much more common in females than males.

It is characterized by attacks of coldness and ischemia of one or more *fingers (or much less commonly, toes, ears, cheeks, etc.), with* numbness, stiffness and actual pain. It is usually symmetrical and may last for minutes or days; it is commonest in winter. With subsidence of the attack, the affected parts usually become red, hot and the seat of severe tingling pain.

The essential pathology is vasospasm.

The symptoms often simulate and accompany early rheumatoid arthritis

Parkinson's Disease (Paralysis agitans):

Charcot first pointed out the resemblance between Parkinsonism and chronic rheumatoid arthritis, especially the *similar* deformities of the hands and feet in the two disorders and the muscular rigidity, tremor and shiny appearance of the face which may occur in both

1. This is occasionally misdiagnosed rheumatoid arthritis because of the semiflexion of the arms and wrists and stiffness and flexion of the head and body.[43]
2. Patients are usually between fifty and seventy, although the onset is occasionally earlier in life
3. Parkisonism is characterized by a rhythmic tremor, mostly in the hands, with a pill-rolling movement of the hands. The tremor appears at rest and is lost by movement (although it is constantly present in advanced cases).
4. The handwriting appears small.
5. The tremor may extend to the legs, lips, chin and tongue.
6. *There appears rigidity of the muscles of the neck, head, spine,* extremities and face.
7. A masklike facies developes
8. Winking is infrequent.
9. It is difficult for the patient to straighten up; he is often bent forward with his head bent forward on the neck, the upper part *of the* trunk slightly flexed, the forearm semi-flexed and the hand in constant pill-rolling motion.
10. All motions are slow
11. There is difficulty in getting started in walking because of the peculiar posture—a propulsive gait.
12. The patient turns *en masse*—the body remaining stiff and straight as a poker (sometimes leading to the mistaken diagnosis of rheumatoid arthritis of the spine).
13. The patient may have a drawing dull pain in the shoulders as an [illegible] as well as muscular weakness

[illegible] ion of fatty tissue; juxta-articular fatty masses may be painful. Joint roentgen-rays are negative, *sedimentation rate is normal.*

n that rheumatoid arthritis is primarily an inflammatory involvement of the synovial membrane with proliferation and secondary involvement of the articular cartilage, whereas in degenerative joint disease, the primary abnormality is in the articular cartilage with fibrillation, pitting, eburnation of the subchondral bone and osteophyte formation at the synovial perichondral junction with little or no involvement of the synovial membrane. In other words, rheumatoid arthritis is an inflammatory process with marked exudation into the synovial membrane and synovial fluid, whereas

Differential Diagnosis of Fibrositis From Rheumatoid Arthritis		
	Fibrositis	Rheumatoid Arthritis
Joint pain	Pain is periarticular, usually worse on awakening, improved following gentle exercise, made worse by rest	Definite joint pain, improved following exercise, improved by rest
Joint swelling	No synovial exudate or effusion	Synovial exudate frequent
Joint tenderness	Absent or very mild	May be marked
Stiffness of joints	Rarely objective, usually subjective only	Commonly subjective and objective
Loss of Weight	Rare	Frequent
Muscular atrophy	Absent	Common
Remissions	Frequent	Uncommon
Constitutional phenomena	Usually absent, usually no loss of weight	Fever, tachycardia, *loss of weight*, etc
Sedimentation rate	Normal, occasionally elevated	*Markedly elevated* in most cases
Anemia	Rare	Common
Non-filament cell count	Normal	Abnormal
Roentgen-rays of joints	*No bony changes*	Definite intra-articular findings
Prognosis	Good, from the standpoint of the joints; recurrences are frequent	Very uncertain, usually poor
Response to therapy	Very variable, occasionally prompt	Slow

type of synovial fluid is common, whereas in degenerative joint disease, effusion is rare and the fluid is non-inflammatory or traumatic. The individual with rheumatoid arthritis has many constitutional features which

me of day at which time the The patient with rheumastiffness, limbers up with

while osteoarthritis never results in bony ankylosis

Osteomyelitis:

Marked pain, swelling and tenderness are noted over the affected area of bone, joint roentgen-ray is usually negative unless the process has extended to the joint. Leucocytosis and septic fever are usually present.

Herpes Zoster:

This may simulate the root pains of spondylitis; diagnosis is possible only when the characteristic vesicular rash has appeared; relief of pain is obtained in some cases by roentgen therapy, injections of pituitrin subcutaneously or iodides (intravenously) Pain is usually unilateral and localized in a single nerve distribution.

Intermittent Claudication:

Cramplike pains in the lower extremities are occasionally mistaken for

or may be absent.

Roentgen examination usually shows calcific arteries. Oscillometric tests show decreased arterial pulsations.

Drug Intoxication:

Arthritic symptoms occasionally follow the administration of some drugs (as arsenic, bismuth, mercury, gold, barbiturates)

Deficiency Diseases:

Scur. about the

Sc ding gums, general tendency to hemorrhages, reduced capillary resistance, subperiosteal hemorrhages, history of diet inadequate in vitamin C; anemia, ready response to vitamin C (ascorbic acid) in adequate doses

Scurvy occurs most commonly under the age of two; arthritis of any form is very rare at this age

Tenderness is usually in the shafts of bones (in scurvy) rather than of the joints

Scurvy may occur in adults, when vitamin C intake has been totally deficient

Vitamin B_1 (thiamin) deficiency.

May produce pains and weakness in the extremities (See discussion of multiple neuritis)

The condition responds to adequate vitamin B_1 (thiamin chloride) therapy

Rickets:

Occurs most often under two years of age

Characterized by pallor, muscular atrophy, swellings in the ends of bones at the epiphyseal line, generalized decalcification of the skeleton

Occasionally confused with arthritis because of disinclination to move the limbs (from hyperesthesia or tenderness)

Heberden's Nodes.—Special mention should be made of the Heberden's nodes which are frequently seen as a manifestation of degnerative joint disease These lesions consist of cystic degeneration and destruction accompanied by firm nodules along the margins of the distal interphalangeal joints of the fingers. At first these nodules may be very painful, but later they subside to leave a firm, hard nodule which frequently causes deformity of the distal phalanx *It has been pointed out that these have a high* familial incidence. These are considered as a manifestation of degenerative joint disease.

Tuberculous Arthritis.—Tuberculous arthritis may at times be mistaken for rheumatoid arthritis. In most instances, however, tuberculous joint involvement is characterized by the *slow, insidious development of a monarthritis,* although several joints may be involved occasionally; involvement of three or more joints is a point against the diagnosis of tuberculous arthritis. Tuberculosis of the spine is the most common form in children, occurring most often between the ages of two and ten. In adults, tuberculosis of the spine seems also to be the most common form, followed by involvement of the hip. The most commonly involved vertebræ are those between the sixth dorsal and fifth lumbar.

In about half or more of the cases, there is a *concomitant pulmonary tuberculosis* In adults, tuberculous joint involvement is most common in the second and third decades. In those with spinal involvement, at least half of the cases develop a paravertebral abscess, which may be at times the only roentgen evidence of the underlying bone disease.

A *family history of tuberculosis,* or a previous personal history of pleurisy with effusion, suppurative cervical adenitis or *fistula in ano* may be of assistance in arriving at the correct diagnosis.

Additional aids in diagnosis of tuberculous arthritis include a biopsy of joint tissue; guinea pig inoculation of synovial fluid; microscopic examination or culture of joint fluid for tubercle bacilli; and a positive tuberculin skin test. The possibility of a tuberculous infection is very strong if fasting synovial fluid has a total leukocyte count below 10,000 cells per cmm, a predominence of mononuclear cells, and a low sugar content [8] The presence of considerable fluid in a joint, or the involvement of numerous (more than three) joints is evidence against the diagnosis of tuberculosis

Symptoms of tuberculous arthritis are not characteristic, except that the disorder usually develops insidiously. Symptoms may include local pain, heat and swelling of a joint, the doughy swelling being due to enlarged synovia rather than to a great amount of fluid; muscular spasm and limitation of motion, muscular atrophy (which may appear early and may be marked); loss of weight; low grade fever and, in some cases, a normal temperature, and local tenderness, which is usually not an early symptom

When the knee joint is involved, pain (insidious in onset) is usually the first symptom, although stiffness may appear as the initial symptom in 10 per cent. Local heat and gross limitation of motion are usually present. In these cases, bacterial examination of the pus (including guinea pig inoculation) from an abscess is a valuable laboratory aid.

[illegible] in the head or [illegible] *often in chil*[illegible] the obturator and anterior crural nerves); muscle spasm; fullness in the groin; tenderness

Characteristic roentgen-ray findings ("ground-glass bones") are diagnostic.

Fairly rapid response to vitamin D therapy is noted.

Paget's Disease:

May cause pains of the extremities and cramps in the leg muscles, occasionally may be mistaken for arthritis.

Usually this is readily diagnosed by the increase in size of the head, marked bowing of legs, and pathognomonic roentgen findings (localized densities).

Occasionally only one bone is involved.

No actual changes in joints occur unless there is an associated degenerative joint disease (osteo-arthritis) which is not uncommon; the pains are chiefly in the bones.

The alkaline phosphatase is elevated in this disease.

Trichiniasis:

Painful inflammatory lesions in muscles occasionally lead to a mistaken diagnosis of arthritis

Usually there is a history of eating poorly cooked pork, sausage, hamburg, etc.

Gastrointestinal symptoms (nausea, vomiting, abdominal pain and diarrhea) preceding muscular pains are usually noted.

Edema of the face, especially of the eyelids, is often found.

Painful, tender, and at times, swollen muscles, are frequently noted

There is a normal white blood count or leukocytosis of moderate degree and almost invariably an eosinophilia (after the second day).

Fever may be marked and is usually septic in type.

Larvæ are often found by examination of muscle tissue.

Postive reaction to intracutaneous injection of trichinella antigen (after the third day of the disease) may be noted; antigen is obtainable from National Institute of Health, U. S. Public Health Service, Washington, D. C.

Acromegaly:

[illegible]

Coarse facial features with thick lips, a large nose and prominent head are commonly seen.

Prognathism (protruding jaw) from overgrowth of the mandible may be present; mastication may be impaired.

Degenerative joint changes, especially involving the spine, are sometimes noted.

Enlargement of the *sella turcica is found in skull x-rays* in 90 per cent of cases.

Headaches and weakness are common.

Visual changes are sometimes noted

Backache, neuralgic pains and paresthesias may occur.

Subacute Bacterial Endocarditis:

Associated arthritis from septic emboli lodging in or around a joint may occur in S B E

The inflammatory reaction usually subsides in several days.

head becomes misshapen or may be entirely absorbed.

Tuberculosis of the spine is the most common form of tuberculous arthritis. The lesions occur most commonly in the bodies of the vertebræ, and in more than half of the cases the thoracic vertebræ are affected The anterior border of the vertebral body is most commonly involved; collapse of

the back, and pains and crying at night, reference of pain at times to the abdomen (along the intercostal nerves); kyphosis; tenderness and muscular spasm over the affected areas and occasionally paralysis of the legs from pressure upon the cord by granulation tissue or cold abscess *Cold abscess formation and paralysis* are the two most common complications. A dorsal abscess may remain localized, or may travel along the anterior aspect of the spine forming a psoas abscess; in some cases, these may point in the thigh

The roentgen findings in tuberculous arthritis are not characteristic. Active early tuberculosis may be present in bones and joints with negative roentgen findings. Soft tissue swellings and increased radiability of the bones may be noted early.

toid arthritis in which the joint involvement seems to be associated with definite focal infection (as dental abscess, sinusitis, peritonsillar abscess, etc). In this condition, only a single or a few joints are involved, as a

process follows shortly after or in association with a focal infection, proper attention to the focus may result in marked lessening of the symptoms and a complete clearing of the process; unfortunately, one does not often see this picture.

Syphilitic Joint Disease.—Syphilitic arthritis is uncommon The Wassermann reaction is positive in most cases and there is a definite

S B E. is diagnosed by history of previous rheumatic fever, cardiac valvular lesion, loss of weight, fever, petechiæ, microscopic hematuria, sweating and positive blood culture for Streptococcus viridans.

Milkman's Syndrome (Multiple spontaneous idiopathic symmetrical fractures):[40]

1. This involves the entire skeleton, flat and tubular bones.
2.

3.

as to direction and location of the lesions.

4. Progression of the process is with periods of intermissions and exacerbations.
5.
6.
7.
8.

9. Crepitus is not found
10. There is tenderness over the skeletal tissues.
11. *X*-ray examination shows bands or zones of increased radioparency. The bone appears as if the entire bony structure were erased at the point of involvement These zones vary from a few mm. to 1 cm in width The bone above and below the transparency looks normal except for a thinning of the cortex and increased striations of the spongiosa and a moderate deficiency of the calcium content The edges of the bones appear irregular. The transparent zones have lost all of their calcium and are composed of structureless supportive or ground substance.
12. The area of calcium deficiency begins in the cortex and gradually
13.

lapping of the fragment may occur

14. The transparent zones increase in number and width
15. There is no attempt at repair and no callus formation, the fragment ends remain irregular and jagged
16. The blood uric acid, urea nitrogen, calcium and phosphorus are normal.
17. The condition is usually misdiagnosed rheumatoid arthritis, sacroiliac sprain, neurosis or osteomalacia. Roentgen examination suggests the possibilities of fragilitas ossium, late rickets, multiple

development, abnormally long gracile fingers and toes resulting in spider-like resemblance, decreased amount of subcutaneous fat producing an appearance of emaciation and accentuating the appearance of length of the extremities.

complete physical examination, blood and spinal fluid Wassermann and the clinical manifestations. *The Wassermann test may be negative in known syphilitic bone and joint lesions*; on the other hand, the finding of a positive Wassermann reaction does not necessarily incriminate syphilis as the cause of the joint discomfort. Too much credence must not be placed upon a negative Wassermann reaction if bone or joint syphilis is suspected, in all these cases, one should *x*-ray the tibia and fibula for additional information.

No pathognomonic roentgen signs of syphilitic arthritis have been reported; suggestive features include erosion of bone and cartilage with bony proliferation and periostitis in a relatively symptomless joint.

Clinical details favoring the diagnosis of syphilitic joint disease include absence of pain, even with marked hydrops, free range of motion, good general health despite a long story of joint pathology, precipitation of the arthritis by a blow (occurring often in syphilitics), other evidence of syphilis, lack of response to salicylates and favorable response to antisyphilitic therapy.

Suspect Syphilis:

1. The occurrence of *joint disease without* heat, *pain* or tenderness must always make one consider neuroarthropathy.
2. *Rigid pupils* and *absent knee jerks* are more important than blood or spinal fluid Wassermann tests in the diagnosis of tabetic arthropathy.
3. Syphilis should always be suspected in all cases of *bilateral painless hydrops of the knees* in children (Clutton joints)
4. Suspect syphilis in any case of so-called "rheumatic fever" which does not respond promptly to adequate doses of salicylates.
5. Suspect syphilis and tuberculosis in a monarthritis or a deforming arthritis at any age

Clutton's joints

Traumatic Arthritis.—Traumatic arthritis is not often confused with rheumatoid arthritis. The *history of injury, monarticular joint involvement* (in most cases) and *absence of systemic manifestations* (*i. e.*, normal T.P.R., normal sedimentation rate and good general health) are of importance in the diagnosis of traumatic arthritis. Bilateral or polyarticular joint involvement is uncommon. In most instances, the traumatic arthritis begins within a week following the injury, although symptoms may not be noted for periods varying from several weeks to six months or more. *Years of pain and limitation of motion may follow a single strain or contusion* even in the absence of evident anatomic changes; severe single injury or repeated mild trauma may result in the picture of degenerative joint disease. One must remember, also, that injury may aggravate an existing arthritis of any type.

Joint effusion may or may not be present, and roentgen examination may be negative in the early stages. Synovial fluid usually contains large numbers of erythrocytes in the early stages and an increased amount of bilirubin resulting from the breakdown of erythrocytes; the icterus index of synovial fluid in traumatic arthritis is usually above 5, while it is almost

The fingers are capable of being distorted into grotesque positions due to marked hypermobility of joints because of relaxation of ligaments.

About 50 per cent of cases also have deformities of joints, expecially of the feet; kyphosis; scoliosis due to inadequate musculature and ligamentous relaxation; high arched palate; prominent ears; marked sexual underdevelopment, and other less important findings.

It is most commonly found in children: tall, underweight and with disproportionately long extremities, who commonly have flat feet, hammer toe or club foot

The condition is transmitted hereditarily as a Mendelian dominant.

BIBLIOGRAPHY

1. Askey, J. M Am Heart Jour, 22, 1, 1941
2. Bamberger, E: Ztschr f. klin Med, 18, 183, 1890
3. Bauer, W. and Engleman, E P.. Tr. A Am. Physicians, 57, 303, 1942
4. Bauer, W. and Coggeshall, H C.: Trans Assn Am Phys, 53, 318, 1938.
5. ——. Med Clin of North. Am, 23, 1173, 1939
6. Bennett, G A, Zeller, J W. and Bauer, W.: Arch. Path, 30, 70, 1940
7. Boas, E. P and Levy, H Am Heart Jour., 14, 540, 1937.
8. Boland, E W: Ann Rheum Dis, 6, 195, 1947.
9. Boland, E W and Carr, W. P.: Jour Am Med Assn, 123, 805, 1943
10. Byron, C S and Michalover, S: Ann. Int. Med., 18, 225, 1943
11. Coggeshall, H C Texas State Med. Jour, 42, 430, 1946
12. Coggeshall, H C and Bauer, W: New England Jour. Med, 220, 85, 1939
13. Coggeshall, H. C. and Hench, P. S Unpublished data
14. Comroe, B I, Chamberlin, G. W and Sunderman, F. W.: Am. Jour Roentgenol, 41, 749, 1939
15. Edeiken, J and Wolferth, C. C: Am Jour Med. Sci, 191, 201, 1936.
16. Ernstene, A C and Kinell, J Arch Int Med, 66, 800, 1940
17. Etter, L E and Glover, L P Jour Am Med Assn, 123, 88, 1943
18. Flind, J. and Barber, H S Quart Jour. Med. U. S, 14, 57, 1945
19. Fried, B M Arch Int. Med, 72, 565, 1943.
20. Futcher, P H. and Southworth, H. Arch Int Med, 61, 693, 1938.
21. Grinker, R R Neurology, Springfield, Charles C Thomas, 1934
22. Halliday, J L: Ann. Int Med, 15, 666, 1941
23. Hargroves, M M, Richmond, H and Morton, R: Proc. Staff Meeting, Mayo Clin, 23, 25, 1948
24. Harris, A W, Lynch, G W. and O'Hare, J P.: Arch. Int Med, 63, 1163, 1939
25. Hench, P S Interstate Post-Graduate Med Assembly, Cleveland Post-Graduate Med, 1, 460, 1947
26. Hench, P. S and Boland, E W. Ann. Rheum. Dis, 5, 106, 1946.
27. —— Ann Int Med, 24, 808, 1946
28. Hench, P S and Rosenberg, E F Arch Int. Med, 73, 293, 1944
29. Herrick, J B. Ann Int Med, 11, 2079, 1938
30. Hollander, L, and Vogel, H R. Penn Med Jour, 43, 1089, 1940
31. Johnson, A C Ann Int Med, 19, 433, 1943
32. Jones, G M, Jr Univ. Hosp Bull, Ann Arbor, 6, 91, 1940

[illegible]

35. Kerr, W J.: Jour. Lab. and Clin Med, 22, 64, 1936

[illegible] 1018, 1943

[illegible] 407, 1935

[illegible]

invariably below this figure in inflammatory effusions. The bilirubin content of synovial fluid may also be elevated occasionally in hemophilia, sarcoma, xanthoma or tabetic arthropathy.

An acute traumatic arthritis, synovitis or periarthritis is usually recognized without difficulty, however, injury may reactivate a previous form of arthritis, or an acute arthritis may develop on a basis independent of the injury. It is well known that traumatic arthritis may result from severe blows or falls, from trauma incident to poor posture or from athletic injuries or occupation. In any form of joint disease, the possibility of a sudden accident or repeated minor trauma must be searched for in the history. Such slight injury [illegible]
door may result in pain in th[illegible]
the introduction of a foreig[illegible]

Characteristics of Traumatic Arthritis

Joint phenomena are usually monarticular.
Symptoms include pain, swelling, crepitus, muscular spasm and limitation of motion
Skin temperature is usually normal
Joint effusion may be present
In most instances, a traumatic arthritis begins within a week following injury although symptoms may not be noted for periods varying from several weeks to six months or more
Roentgen examination may be negative in the early stages and may reveal [illegible]

carbon dioxide or contrast medium
[illegible]

arthritis, or an acute arthritis may develop on a basis independent of the injury
Differentiation of a sprain [illegible]

joint picture of a degenerative joint disease

Lymphogranuloma Venereum.—Lymphogranuloma venereum occurs more commonly in negroes The Frei test is specific for this disease. The joint manifestations are usually milder than rheumatoid arthritis and respond more readily to rest; the process responds to sulfonamide therapy

Report

31, 1945.

46. REIFENSTEIN, E C, REIFENSTEIN, E C, JR and REIFENSTEIN, G H Arch. Int Med, 63, 553, 1939
47. ROSE, E and PILLSBURY, D M Ann Int Med, 12, 951, 1939
48. RYNEARSON, E H and SACASA, C. F.: Proc Staff, Meet Mayo Clin, 16, 353, 1941
49. SCHÖNLEIN: Allg u. spec Therap, Herisau, 2, 1848, 1837
50. SHORT, C L New England Med Jour, 236, 383, 1946
51. SLOCUMB, C. H: Proc Mayo Clin., 15, 683, 1940
52. ——— Jour. Lab and Clin. Med, 22, 56, 1936
53. THOMPSON, T. C Med Clin. North America, 21, 1785, 1937
54. VAN HAZEL, W.. Jour. Thor Surg, 9, 495, 1940
55. WEISS, E Ann Int. Med, 26, 890, 1947

The arthritis with lymphogranuloma venereum is usually a chronic process, with acute flare-ups and a tendency to relapse. Polyarticular involvement is the rule. Migratory joint involvement or generalized arthralgia may occur. The *joint involvement may persist for weeks or* months without destruction of bone or cartilage. The swelling is usually confined to the periarticular tissues.

Other evidence of lymphogranuloma venereum which may be found

arthritis with such treatment.

OTHER DISEASES SOMETIMES MISTAKEN FOR RHEUMATOID ARTHRITIS

A list of additional conditions which, under certain circumstances, might be confused with rheumatoid arthritis is given.

Bursitis (subdeltoid, trochanteric)
Tendonitis (supra spinatus)
Neuritis, peripheral
Allergic Reactions
  Serum Sickness
  Drug Sensitivity
Primary Osteoarthropathy
Endocrine Disturbances
  Myxedema
  Acromegaly
  Hyperparathyroidism
Infections
  Subacute Bacterial Endocarditis
  Trichinosis
Circulatory
  Raynaud's Syndrome
  Buerger's Disease
Vitamin Deficiency
  Scurvy
  Beri-beri
  Rickets

Mechanical Skeletal Disturbance
  Paget's Disease
  Scalenus Anticus Syndrome
  Cervical Ribs
  Neoplasm, primary and secondary
  Neuralgia (Radiculitis from spinal osteoarthritis)
Other
  Leukemia
  Polycythemia
  Hemophilia
  Sarcoidosis
  Scleroderma
  Dermatomyositis
  Milkman's Syndrome
  Arachnodactylia
  Parkinsonism
  Osteochondritis Dissecans
  Osteomyelitis
  Ochronosis
  Shoulder-Hand Syndrome
  Hysteria
  Malingering

Since many of the above conditions are described adequately in other chapters of this volume, only those not discussed elsewhere will be described briefly on the following pages

Acute Disseminated Lupus Erythematosus and Periarteritis Nodosa.— Clinically, these two conditions are considered together because they frequently cannot be differentiated from one another even *at postmortem* examination. Arthritis, among other systemic involvements, is generalized

CHAPTER 15

TREATMENT OF RHEUMATOID ARTHRITIS

By Ralph H. Boots, M.D.

The greatest arthritis problem confronting the practitioner of medicine i the patient with rheumatoid arthritis. Only too frequently the patient receives no treatment until the disease is well advanced. In comparing results of therapy, there seems to be common agreement that the earlie treatment is begun, the better the outcome. Unfortunately, because o the insidious development of this disease over a period of weeks, month or years, the patient may have his disease for a long period before it i recognized, so the first requisite to good therapy is the ability to make a early diagnosis



Fig 73 —Rheumatoid arthritis Note fusiform swellings of proximal interphalangea joints

The purpose of this chapter is to outline the important therapeuti measures now in use or which have been used in rheumatoid arthriti

Until the true etiology of rheumatoid arthritis is known, the treatment m remain empirical. Since a specific treatment is not yet available, it i important for the physician to use the measures which have proved most helpful through years of clinical trial even though they are empirical an often inadequate. Today we speak of "management" of rheumatoi

and frequently resembles rheumatoid arthritis. In that these conditions involve the arterioles and the smaller vessels of the vascular tree, the symptoms of the disease are wide-spread and involve many systems. For this reason, these patients may have asthma, polyserositis (pericardium, pleura and peritoneum), myocarditis, valvular disease, chronic nephritis

seminated lupus erythematosus with or without skin lesions. Chronic

Differential Diagnostic Features)

Peripheral Neuritis:

Neuritis may produce sharp burning pain in the distal portion of the extremities, chills, fever, general malaise, exaggerated tendon reflexes (in an early stage), tingling and numbness in the hands and feet,

vitamin deficiencies, leukemia and diphtheria.

Deformity of the hands produced by neuritis is occasionally incorrectly diagnosed as arthritis.

Diabetic neuritis is rare under the age of forty.

Chronic arsenic poisoning may be associated with painful joints in the extremities.[16]

Brachial neuritis may simulate subdeltoid bursitis or arthritis of the shoulder joint

may result from dyspituitarism [19]

It is usually considered to be secondary to chronic suppuration in the lungs, intrathoracic tumors,[18] congenital and valvular heart disease, chronic lesions of the liver and intestinal tract, polycythemia vera or

wh

thelioma of the pleura Six of the 24 had pulmonary osteoarthropathy with dependent edema of the extremities, and in four additional cases

arthritis This includes the proper use of all the therapeutic agents which will ameliorate its symptoms These agents are: (1) properly balanced rest and activity, (2) salicylates and phenylbutazone (Butazolidin), (3) physical therapy, (4) psychotherapy, (5) measures to prevent or correct deformities, (6) treatment of the anemia with iron or transfusions, (7) attention to adequacy of diet and vitamin intake, (8) chrysotherapy, (9) cortisone, hydrocortisone, or adrenocorticotropic hormone therapy, (10) roentgen therapy (of value only in rheumatoid spondylitis), and (11) removal of proven intercurrent (focal) infections.

The beneficial action of most of these is due to the improvement of the patient's general health, permitting him to develop a remission Salicylates

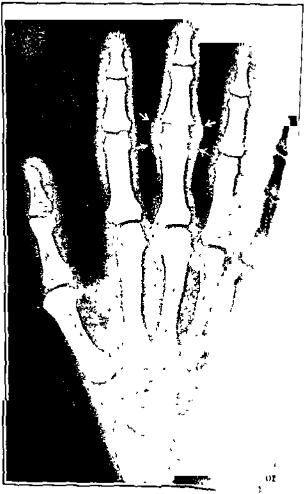


FIG 71 —Early *rheumatoid arthritis* showing very w[illegible] at the proximal interphalangeal joint of the middle f[illegible] joint space

there was not only the pulmonary osteoarthropathy, but also joint findings compatible with the diagnosis of rheumatoid arthritis. Others showed findings of frozen shoulder, or other types of rheumatic disease.

In these cases the pulmonary osteoarthropathy subsided after surgical removal of the tumor, but recurred along with recurrence of the chest lesion in three cases. Polley concluded that fibrous mesothelioma of the

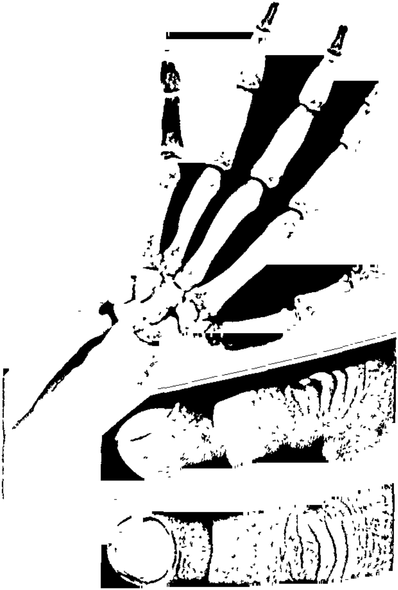


Fig 72 —Hippocratic finger and roentgenogram of hand showing the characteristic features of hypertrophic osteo-arthropathy (Campbell *et al*, Proc Mayo Clin)

and phenylbutazone are of value in *relief of pain* but do not induce a remission. Of particular value are those agents which in themselves are capable of producing a remission (even though temporary) in a sizable percentage of patients. So far the only ones of practical importance which may accomplish this are the hormones—adrenocorticotrophic hormone (ACTH), hydrocortisone (Compound F) and cortisone (17-hydroxy-



narrowing of joint spaces, erosion of bone and ankylosis

11 dehydrocorticosterone: Compound E)—and the gold compounds which contain a sulfhydryl group.

Success in treating rheumatoid arthritis lies in the ability of the physician to use a *combination* of the above agents. The revolutionary approach to the problem of etiology and treatment of rheumatoid arthritis by Hench,

Kendall, Slocumb and Polley[15] in 1949 has so stimulated thought and research of this disease that most physicians are now familiar with the possibilities of benefitting the patient. In approaching the problem of treatment, several important facts should be borne in mind:

1 *Do not promise the patient with rheumatoid arthritis a cure!* It is only fair to explain to the patient that once the hyaline cartilage over the ends of the bones has been destroyed, it does not regenerate, although there may be some attempt at repair by connective tissue or fibrocartilage formation.

2. The patient should be told that there are 7 chances in 10 that he or she will obtain marked improvement, and an additional 2 chances in 10 that some improvement will follow; the family should be warned, though not pessimistically, that in about *5 to 10 per cent of cases, the arthritis continues or progresses despite all treatment.*

3 The physician is justified in *manifesting a degree of optimism* and confidence to the patient concerning the outcome of his particular illness. However, such optimism must be genuine and not simply overenthusiasm with a promise of rapid cure.

4 The physician must stress the point that with our present knowledge, the *treatment may* in all probability *require several years,* and not weeks or

varying with the conditions which are met in that particular patient.

The physician is often confused by the availability of a multitude of measures which may be employed for the patient with rheumatoid arthritis He must stress to the patient that no pill or capsule, as yet known, and no combination of oral medications will cure all patients with the disease Even though cortisone and ACTH are now readily available, and have proven themselves to be very valuable in therapy, *they do not cure* and are certainly *not* to be recommended for all patients. He must

severe Exacerbations may accompany marked changes in weather, acute respiratory infections, emotional crises, periods of overactivity, menses, or may follow childbirth, while partial or almost complete remissions may occur during pregnancy or jaundice. *The tendency to spontaneous remissions in rheumatoid arthritis must always be considered in evaluating the*

the process. In order to obtain the best results in the treatment of rheumatoid arthritis, it seems reasonable that we must treat the whole individual including both his body and his mind.

he rest, vacation away from the cares of the household, and the value obained from heat, massage and hydrotherapy. (See Chapter on Spa Therpy.)

(m) In chronic arthritis, general heliotherapy may be of tonic value to mprove the appetite and sense of well-being. Too much exposure to sunhine or ultra-violet irradiation in patients with acute rheumatoid arthritis night produce increased activity of the joint process. No one has ever lemonstrated that sunlight has any *specific* beneficial effect upon the course f rheumatoid arthritis.

(n) Occupational therapy *is always* of value in keeping the patient's nind occupied so that he does not constantly think about his illness, the vork should be interesting and absorbing *for the patient*—the "he man" vith arthritis does not usually like to weave mats! The tasks should be lesigned to encourage use of weakened muscles and stiffened joints—see also under Occupational Therapy.

Diet in Rheumatoid Arthritis

1. In most cases [illegible]
2. [illegible]
3. [illegible] fresh green vegetables, butter, cream and eggs as desired, and any additional foods chosen by the patient provided that these contain a [illegible]
4. [illegible] of vitamin C
5. *There is no good reason why any food or class of foods should be restricted from the diets of these individuals* unless such articles cause gastrointestinal upsets or the patient is allergic to such foods

(4) Diet in Rheumatoid Arthritis. One of the [illegible]

[illegible] any specific [illegible]

An important consideration often forgotten in the planning of treatment is the enlistment of the patient's own efforts to help himself. Arthritic patients all too often are willing to sit back and wait for the doctor to produce a cure. The patient must be directed to plan his day to include proper exercises, balanced by adequate rest, various self-applied home physical-therapeutic measures, proper selection of diet, and diversional activities, as well as simply taking prescribed medicines at the proper time. If the patient

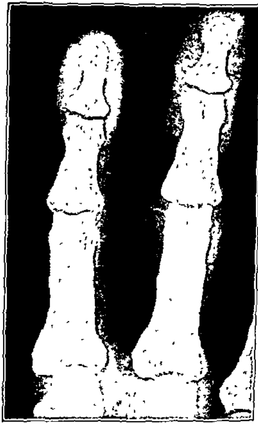


FIG. 76.—Early *rheumatoid arthritis*. Middle finger is at right Note soft tissue swelling (fusiform) at proximal interphalangeal joint and narrowing of this joint space, *with several small punched out areas of bone here, so often seen in rheumatoid arthritis*

is thoroughly convinced of the importance of his own part in the treatment, his cooperation will be assured. The patient thus is made an "active partner" rather than a passive recipient of therapy.

Treatment will vary with the stage of the disease process. In the very early stage of the arthritis, it is often possible to obtain marked improvement or arrest of the joint symptoms In those individuals in whom the disease, although of several years duration, is now relatively quiescent, a regimen of ... and general ... ct ... it-

ment during the active stage of the disease.

amount of fresh green vegetables, fruits, milk and meat, which seem more essential.

In addition to a well-balanced diet with an adequate caloric intake, one should add vitamin concentrates as capsules or other forms; *thus far, there seems to be no specific effect upon the arthritis which is achieved by any one particular vitamin.* Nausea, vomiting and the dangers incident to a persistently elevated blood calcium may follow the prolonged use of massive doses of vitamin D.

[illegible]

even though it is kept cold in the refrigerator. In these individuals, Maltine with cod-liver oil may be tried.

If the patient drinks a full glass of orange juice daily, it does not seem necessary to add extra ascorbic acid (vitamin C) to his diet. In some of these individuals with poor appetites, vitamin B complex (capsules) often aid in improving the appetite and the general sense of well-being. If these are not tolerated, one may try thiamin chloride alone (5 mg. b i d.), or in combination with riboflavin (1 to 2 mg. per day) and niacinamide (100 mg per day). *Great care must be taken not to overload the rheumatoid arthritic* [illegible]

(5). **Relief of Joint Pain.**—(a) **Salicylates and Related Analgesics.**—The physician must attempt to relieve pain in the patient with rheumatoid arthritis. *Pain prevents proper rest, appetite, sleep and exercise, which are important factors in the recovery process.* Peripheral circulation and muscle function may be decreased by persisting pain, possibly through nervous imp[illegible]
ical [illegible]
lates [illegible]
rheumatoid arthritis, aspirin (acetyl salicylic acid) and sodium salicylate are the most commonly used preparations. The average patient is much more comfortable taking dosages of 0 3 gm. (5 grains) to 0.6 gm. (10 grains) after meals and before bed. If these medications cause gastric irritation, enteric coating of the salicylate preparation will often give relief. Enteric coated aspirin in five or ten grain tablets is now becoming more available, and has been tried in several clinics on large numbers of rheumatoid arthritic patients. Such a preparation is better tolerated over a long period than uncoated forms, but does not act as promptly in relieving morning stiffness.

Combination of salicylate with sodium para-amino benzoate (PABA) has been used but in most cases has little advantage over salicylates alone.[18] Sodium gentisate has also been tried, but has such transient analgesic action that it holds no advantage over salicylates in most cases. [illegible] Five grains [illegible]

[illegible] If this [illegible] rated,

then think of cortisone.

The patient must not expect constant improvement without interruption; the response to therapy will be variable from week to week and minor setbacks will occur in most patients.

MEASURES OF PROVED VALUE

Over a period of years certain therapy for rheumatoid arthritis has stood the test of time and has been shown as of *real value*. It is the endeavor of the author to separate such treatment from methods of doubtful or little value.

(1) **Adequate Mental and Physical Rest.**—(a) A change in environment may be required hospitalization; spa; vacation, etc

(b) A period of several weeks or months of rest in bed may be necessary in cases of active generalized rheumatoid arthritis

Measures of Proved Value in Rheumatoid Arthritis

1 *Rest* of the body: mentally and physically.
2. Rest of the involved joints, with *splinting* of the joints to prevent deformity, when indicated.
3 *Physical therapy* (heat, massage and special exercises) and *occupational therapy*.
4 A sensible *nutritious diet* containing adequate calories, vitamins and minerals and anticonstipation factors
5 *Relief of pain*: splinting of affected joints, rest, physical therapy, analgesics when required (especially *aspirin* and often phenylbutazone), and sedatives and hypnotics if needed
6 *Avoidance of cold, dampness* and *drafts*
7 *Psychotherapy*, and proper attention to maladjusted human relationships leading to emotional reactions, especially anxiety and resentment
8 *Orthopedic measures* including supports, good posture, elastic bandages, braces when indicated, proper shoes, corsets, plaster casts, manipulation of joints and orthopedic surgery, if required (but only under expert supervision)
9 [illegible]

[illegible]

10. [illegible]

[illegible] d that gold when [illegible] parenterally, will [illegible]

to a remission.
13 [illegible]

14 [illegible]

of the above measures.

There is little justification for the intravenous use of salicylates in the patient with rheumatoid arthritis; most of the patients tolerate oral medication if prescribed intelligently.

(b) **Phenylbutazone (Butazolidin).**—This new drug has appeared to be a very effective agent for the relief of pain in many forms of arthritis, and consequently has usefulness in rheumatoid arthritis, *but should be prescribed with caution!* Both phenylbutazone alone and in combination with aminopyrine (Ergopyrine) have now been used in treating hundreds of cases of various types of arthritis.[21 22 24]

At the time of this writing, several conclusions seem justified: (1) Phenylbutazone is a highly effective analgesic. (2) It is as effective alone as with aminopyrine. (3) It is frequently toxic, producing edema, skin rash, gastro-intestinal upsets, or even blood dyscrasias in a sizable pro-

ACTH.

Phenylbutazone is available in enteric coated tablets of 100 or 200 mg. each, or in solution for intramuscular use. The usual dose for an adult is 200 mg three times daily, but some patients can be relieved of pain on 100 mg t.i d., whereas others require as much as 200 mg. q i d for relief. The degree of analgesia produced is sometimes so great that patients receiving the drug as an adjunct to cortisone therapy have been able to reduce the required amount of cortisone to half the previous dose.

The greatest disadvantage of phenylbutazone in therapy is the rather high incidence of toxicity from its use. There is a definite tendency to produce sodium retention, especially in patients over 60 years of age, in cases

sation

promp

abdominal discomfort are also frequently encountered, in spite of the enteric coating of the tablets.

usually respond to withdrawal of the drug, and, in severe cases, to transfusion or a short course of ACTH.

effect, since the degree of swelling usually does not decrease appreciably with its use

At the present time phenylbutazone is still being intensively studied in

its full potential value and

ere can be no question that

ther this is sufficient over a

toxicity remains to be de-

It is certainly not a new "wonder drug," but seems to be a useful therapeutic adjunct[21 22 24]

(c) If the patient must continue his occupation, daily rest periods should be instituted.

Almost all of these patients complain of fatigue. *Fatigue is one of the greatest contributing or precipitating factors of all disease.* The elimination of fatigue will do much to restore the mental and physical endurance of these individuals; this is a very difficult thing to accomplish in some cases.

A period of several weeks or months of rest in bed ...

in bed in any position which he deems best.

The patient with rheumatoid arthritis is a sick individual. *He has a generalized disease, not only a local disorder of his joints.* As in pulmonary tuberculosis, where few physicians doubt the value of rest in bed during the active stages, a proper amount of rest in bed during the active stages of rheumatoid arthritis will usually pay large dividends. During this period, ankylosis, contractures and muscular atrophy are prevented by physical therapy with prescribed bed exercises, and proper orthopedic care. As a general rule (except in acutely inflamed joints), each joint should be moved through as full a range of painless motion as possible *at least* several times a day to prevent the development of adhesions. Properly administered massage (of the muscles between the joints) and supervised active and passive movements will help maintain muscular tone and prevent extensive muscle atrophy and contractures.

If there is severe pain or beginning contracture, the affected joint should be splinted for limited periods during the day or night, in an effort to prevent trauma to joints and to promote muscular relaxation. Methods of splinting and positions to be used are discussed ...

the patient is ambulatory and his arthritis is not severe or disabling, he should be encouraged to work if this is a non-traumatizing form of activity. If the individual is worn out and tired, he must rest for a period varying from ten to twenty hours a day, but special exercises must be prescribed ...

range possible in *all* joints, but should not be severe enough to produce pain which persists for periods of more than one hour. They should never be so strenuous as to tire the patient unduly ...

tuberculosis, and depends upon the response of the patient. (2) If possible ...

(c) **Sedatives and Hypnotics.**—Sometimes the joint pain is so severe that codeine 0.015 gm. (¼ grain) to 0.03 gm. (½ grain) is necessary. The administration of morphine is to be avoided. This is *treatment for a* chronic disease and use of narcotics should be minimized. However, in an occasional elderly patient with rheumatoid arthritis, the use of oral demerol for relief of pain seems justified.

needed and should be prescribed unless the insomnia is due to pain.

(7) **Psychotherapy.**—Individuals with rheumatoid arthritis are usually *introspective, fretful, lack confidence in themselves (and occasionally in* their physician) and are often seemingly unreasonable; *psychotherapy*, whether used consciously or unconsciously, is *absolutely essential to the proper handling of these patients*, who are especially amenable to suggestion

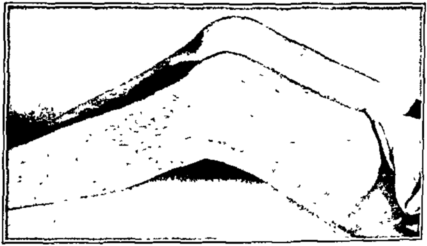


FIG. 79.—Flexion contractures of knees in a patient with rheumatoid arthritis

Psychotherapy is an extremely potent weapon in the armamentarium of

ment. The effect of any hypodermic or intramuscular needle, an optimistic approach by the physician, and the patient's knowledge that his physician [illegible] of treatment available if the first type is unsuccessful all [illegible] should be pointed out to the [illegible] ility of stiff joints, even slight decreases in the sedimentation rate or increases in the hemoglobin content, etc. Minor flare-ups or increased pain can be "blamed on the weather" *Never blame the patient* as this adds to his depression.

The arthritic patient must be questioned carefully about his intimate relationship with members of his family, his friends and business associates. One must try to expose to the patient his resentments, conflicts or actual

may then be allowed to increase the frequency and duration of his periods of activity. (4) Ambulation and general rehabilitation of the arthritic patient should be closely supervised. The *optimum exercise-rest balance* must be maintained throughout. This portion of the treatment is discussed fully in the chapter on Physical Therapy in Arthritis.

Rest in Bed Should be Insisted Upon in:
1 [illegible]
2 [illegible]
3 [illegible]
4 [illegible]

The necessary period of rest in bed varies with the individual In some, a stay of one or two weeks may be markedly beneficial, while in a few severe cases from four to six months may be required to produce any marked benefit When rest in bed is absolutely impossible, periods of rest upon a couch [illegible]

Even in the subacute stages, limitation of weight bearing will usually produce added improvement *The climbing of stairs is to be avoided whenever possible by those individuals with arthritic involvement of the knees, feet or hips.*

If Prolonged Rest in Bed is Necessary:
1 [illegible]
2 [illegible]
3 [illegible] satisfactorily
4 The patient must not lie in bed like a lump of putty, *but should have a firm mattress and a non-sagging spring.* He should be taught the proper positions of rest for his extremities to avoid contracture deformities, *muscle setting exercises,* and as soon as tolerated, *graded active exercises*

Weight Bearing is Inadvisable and not Permitted if:
1 Weight must be borne on acutely inflamed or hot joints
2 There is extreme muscular atrophy of the legs
3 The knees are fixed in flexion or the legs are not in good alignment.

In patients with mild rheumatoid arthritis in whom prolonged rest in bed may not be necessary, a period of ten to twelve hours of rest in bed each night should be prescribed, as well as from one to two hours each afternoon.

hatreds and his fears. (See also Chapter on Psychogenic Factors in Rheumatic Diseases.)

(8) **Cortisone, Hydrocortisone and ACTH.**—Following the original report of Hench and his colleagues[15], there was a period when most investigators viewed these hormones as "research tools" and hesitated to recommend their general use in rheumatoid arthritis. Now that many patients have

widely used special treatment, cortisone, hydrocortisone or ACTH may be more depended upon to produce a remission. The technique for cortisone, hydrocortisone, and ACTH therapy, contraindications, and the untoward reactions of these hormones are described in detail in a later chapter. Hydrocortisone is reported to be equally or even more effective than cortisone orally.[4] (See Chapter 17.)

A trial with cortisone or ACTH therapy seems justified in patients with advanced rheumatoid arthritis in whom the disease has been present for a number of years and who have considerable pain and deformities. The expense of the hormone must also be considered. On the other hand, there would seem to be a question of choice in the patient who has early rheumatoid arthritis. The choice lies between (*a*) hormone therapy, (*b*) a regimen of rest and general hygienic measures hoping for a spontaneous remission which occurs in a definite percentage of cases, and (*c*) this latter regimen plus gold therapy. It seems practical to treat patients who have had the disease for less than one year with gold therapy rather than starting in with hormone therapy. In patients with severe rheumatoid arthritis who

peutic agent for relief. Reports of the combined use of cortisone and gold have been conflicting, some investigators claiming there is a mutual antagonism of these two agents. (See Chapter 16.)

Intra-articular Hydrocortisone injection has proved a useful local adjunct in the management of rheumatoid arthritis,[20] and is described in detail in another chapter. (See Chapter 18.)

(9) **Chrysotherapy.**—Gold sulfhydryl compounds are widely used in the treatment of patients with rheumatoid arthritis and will be discussed in detail in a later section. *In the author's clinic at the Presbyterian Hospital*, we have used such therapy for the past thirteen years and are convinced that in a high percentage of cases it can cause remissions in rheumatoid arthritis. Chrysotherapy was introduced and popularized by Jacques Forestier.[9]

More recently Forestier[10] recommended the use of a copper sulfhydryl preparation (cupro-allyl-thio-urea-benzoate of sodium). He claims that it does not have the toxic effects of gold compounds. The beneficial action of copper sulfhydryl preparations has not been confirmed by other investigators.[21]

Adams and Cecil[1] concluded that "gold salts, if administered during the first year of rheumatoid arthritis, increase and accelerate the appearance of remissions." They obtained remissions in 66 per cent of their one

If the long night rest produces too much stiffness, it may be broken by short "limbering-up" periods of activity as needed. If the patient is fatigued despite this program, he may require more time in bed, but sometimes his fatigue may be due to *worry* or *mental unrest* in which case additional bed rest will help but little. Even as the patient improves, he should continue to obtain, if possible, his afternoon rest, rest after dinner in the evening, and a good eight hours in bed each night.



... years, who
...ith marked
The small
...r advanced
...e articular
the meta-

(2) **Rest of the Involved Joints.**—(a) Marked improvement may be noted if acutely inflamed, tender joints are placed in positions of physio-

...zed (when-
...ologic rest
...ns are de-
...ities.

arthritis, has now died down to a trickle. Several reasons for the abandonment of sulphur therapy are apparent:

(a) Freyberg and his associates,[11] by carefully controlled experiments, exploded the theory that there was a disturbance of sulphur metabolism in the arthritic patient, or that there was a biochemical need for sulphur.

(b) The rising popularity of gold therapy literally pushed sulphur off the arthritic map

It is apparent following Freyberg's work that sulphur is not indicated in arthritis due to any evident lack of sulphur or disturbance in sulphur metabolism in the arthritic patient; however, Freyberg admitted that it is possible that sulphur may have a non-specific beneficial effect.

One of the most significant facts concerning the use of sulphur in arthritis is that, at present, we know of no large arthritis clinic associated with a teaching hospital which is using sulphur therapy routinely in the treatment of any form of arthritis. Warm mineral springs containing large amounts of sulphur have long been used as a part of the arthritic armamentarium



material

tion

Remarkable curative effects of such baths have been claimed. It is doubtful if the sulphur content of these warm waters plays any rôle in this improvement, except perhaps a psychotherapeutic one. It is true that many patients will eagerly submit to warm mineral baths while these same individuals would rebel at the thought of mere warm water baths as a part of their physical therapy

(3) **Bee Venom.**—Bee venom, either in the form of injectionable venom or actual bee stings, has been recommended periodically by scattered workers ... It has often been stated that

as one of his patients who obtained no benefit from bee venom therapy

should be discarded as soon as the acute inflammation or threat of deformity has subsided. (See chapter on Prevention of Deformities.)

When Weight Bearing is Begun.—If the lower extremities have been in-

habilitation of the Arthritic Patient.)

3. **Physical and Occupational Therapy.**—(a) Physical therapy, and in a broader sense *physical management*, in arthritis is one of the most important parts of a comprehensive program of treatment. A completely planned

preventing the deformities of arthritis and particularly can inhibit the marked muscle wasting which is so common in this disease. *Physical therapy is, in most cases, the most neglected useful method of treatment for rheumatoid arthritis.*

(b) This special therapy should be dispensed with care, under the direction of a trained physician. The cooperation of a well-trained physical therapist is necessary, both to administer the special treatment, and to instruct members of the patient's family in the proper use of massage, exer-

application of heat. In those individuals whose symptoms are aggravated by heat, the intensity and duration of application should be decreased. (See also chapter on Physical Therapy in Arthritis.)

(d) In the acute stage of an arthritis, *do not employ* diathermy, short wave diathermy, or micro-wave therapy, as the symptoms are frequently aggravated by such procedures. Prolonged hot baths are too debilitating for the severely ill arthritic patient.

very effective.

(f) Under-water therapeutic exercise is very effective in stimulating

possible out of the water. Aided by the buoyancy of the water, extension of affected joints may be performed in marked comfort. *Buoyancy is present*

Numerous toxic symptoms have occurred from this form of treatment Certainly, it can be said that the beneficial results from this form of therapy are so poor that it has been discontinued as of no value.[17]

(4) **Chaulmoogra Oil.**—The use of chaulmoogra oil in arthritis is entirely empirical McIlhenny,[23] orthopedic consultant to the National Leprasarium at Carville, Louisiana, noted in 1926 that the "secondary infectious type of arthritis" (probably rheumatoid) was not seen in patients afflicted with leprosy. In an effort to explain why lepers did not develop rheumatoid arthritis, he concluded that chaulmoogra oil (which they received) was responsible for its prevention.

paper of McIlhenny appeared in 1931, there have appeared to date very few reports on the use of chaulmoogra oil from any of the large recognized arthritis clinics in this country

(5) **Cobra Venom.**—Venom from several species of snakes, including the cobra, pit viper, water moccasin, and copperhead, has been employed by various workers in the treatment of arthritis and allied conditions Most qualified workers at present find a lack of scientific evidence for any of these venoms in the treatment of rheumatic conditions[31] None of the venoms have an established place in the treatment of arthritis.

(6) **Prostigmine or Neostigmine.**—Trommer and Cohen,[32] in 1944, reported prostigmine as beneficial in releasing the muscle spasm in rheumatoid arthritis and allied conditions An extensive investigation of this was made by Balboni, Hollander, and Kydd,[2] in 1946, and they were unable to confirm this work and concluded that "we do not recommend the use of prostigmine in the treatment of rheumatoid arthritis "

(7) **Antibiotics and Sulfa Compounds** —In view of the possible connection between rheumatoid arthritis and hemolytic streptococcus infection, both the sulfa group of drugs and penicillin have been tried Results have been most disappointing.[3] Streptomycin, aureomycin, and chloramphenicol have likewise had little effect on the course of rheumatoid arthritis

Brown *et al.*[7] have recommended small doses of terramycin for treatment

no therapy "

(8) **Bogomolets Anti-Reticular Cytotoxic Serum (ACS).**—Various investigators in Russia have claimed that small doses of an "anti-reticular cytotoxic serum" greatly stimulate reticulo-endothelial cells, while large doses block these cells The anti-reticular cytotoxic serum (ACS) is usually prepared for use in humans by immunizing an animal (horse, donkey, sheep, goat) with antigen taken from the spleen and rib marrow of a human cadaver (in cases who have had no infectious diseases) not later than six to ten hours after death The serum as used in this country, for the most part, was pre-

in any water, whether it is at the mo
made pool or bath tub. Hydrotherap
rheumatoid arthritis because the
fatigued.

(g) Hot paraffin applications are often useful in the treatment of chronic arthritis of the hands and wrists. For detailed technic, refer to the chapter on physical therapy. *Paraffin applications often aggravate acutely involved joints.*



FIG. 78.—Long-standing *rheumatoid arthritis* in a woman aged sixty years. There are extensive changes involving the articular surfaces and adjacent portions of the bones of the knee joint. There are also changes in the soft tissues. On the mesial side of the knee joint, there are some hypertrophic changes. Note the marked narrowing of the joint space, indicating destruction of hyaline cartilage.

(h) Any object which has been heated to a higher temperature than its

minutes several times a day, and the maximum heat tolerated comfortably

pared by Dr. Goldblatt and his associates, the only difference from the Russian serum being that rabbits were used instead of horses. This serum [illegible]

perience of all workers seems to have been well summarized in the paper by Freyberg and associates,[26] which indicated that only a very small percentage of patients were benefited, and it was impossible to determine how many of this small group were having a spontaneous remission.

There is little or no objective evidence of its value in the treatment of rheumatoid arthritis.

(9) **Iodides and Arsenicals.**—There is no specific or unusual benefit to be derived from either of these, although their use has been popular at various times

(10) **Spinal Pumping.**—In 1935 Speransky, assuming arthritis to be a neurodystrophy, applied spinal pumping (*i.e.* repeated extraction and reinjection of spinal fluid) to patients with "chronic arthritis". His results, as those of the Gillmans[12] are questionable because of failure to differentiate the types of arthritis treated. Others[5] have reported on this unorthodox method with unconvincing results. The procedure is not without danger, as one death occurred.[12] This treatment seems neither rational nor acceptable.[14]

(11) **Pregnenolone and Other "Mysteroids."**—Following the discovery of the effect of cortisone and ACTH on rheumatoid arthritis, a rash of optimistic reports appeared in the literature on the supposed effectiveness of delta-5 pregnenolone, *21-acetoxypregnenolone, testosterone,* desoxycorticosterone with ascorbic acid and other steroids in treating the disease. Continued observation, and controlled studies[16, 22] have shown that none of these agents offer any consistent help to the arthritic patient. Since *many little known steroid compounds were being tried* therapeutically, Hollander[19] grouped them under the term "Mysteroids," and found none of them to have any significant effect on the inflammation of the rheumatoid arthritis joint as measured by the intra-articular temperature.

Ragan, et al[25] *have recently stated: "Every year, since* the older of us believed we had reached the age of medical reason, and probably long before that, new therapies have been advocated for the treatment of rheumatoid arthritis. The list of suggested therapies has reached astro*nomical proportions, and much of the time of the men* interested in this field has been spent in refuting or confirming such allegations. Some workers have never been able to confirm any of them."

Investigation is going on in many arthritis clinics on the effect of new *treatments for arthritis Some drugs under investigation are directed simply* [illegible]

treatments are constantly appearing, and old discarded treatments are sometimes pulled out of their dusty oblivion for another brief fling. Until new treatments are adequately studied on a large, controlled series of patients, *with objective evaluation of results,* it is best to use the measures *of proved value, even* though they may seem prosaic and dull both to the physician and the expectant patient. An optimistic attitude is necessary

treatment of many subacute and chronic painful, inflammatory conditions,

diathermy is not used in acute arthritis. In fact, *diathermy is not essential in the treatment of rheumatoid arthritis.* In chronic arthritis, subjective and

more relief when *hot fomentations, poultices or mudpacks* are used *over the affected joints* for twenty or thirty minutes, 3 or 4 times per day. This may

possible, the blankets are soaked in the hot water every several minutes and reapplied as often as necessary (to prevent cooling of the fomentation). This application and reapplication of the hot wet blanket or towel, wrapped around the joint, is continued for twenty to thirty minutes, 3 or 4 times a day When hot fomentations are not available (which is rare), one may employ a heating pad, hot water bottle, home-made baker, infra-red lamp or other source of heat *That form of heat which is most analgesic should be continued.*

(k) As an acute arthritis subsides one may begin effleurage (very light massage) and increasing exercises in bed (without weight bearing) Too much motion may cause further joint irritation, prolonging the process, but too little motion in this and later stages will usually encourage the formation of deformities, and will certainly produce muscle weakness.

Occasionally, it may be impossible to exercise joints because of marked pain, in such instances, if muscle setting exercises cannot be performed, faradic or *sinusoidal currents* may be used to produce relatively painless contractions of muscles, thus reproducing the effects of active exercise and preventing atrophy of adjacent muscles.

(l) *General Physical Management.* In resting the involved joints, one must include instructions as to reduction or possibly complete *avoidance*, if possible, *of postural, static, recreational or occupational trauma* The static trauma from marked obesity or poor posture may be lessened by decrease in weight, supports (elastic bandages, corrective shoes with properly placed pads or metatarsal bars, corsets, braces, etc) and postural exercises. One must be very careful in advising arthritis patients to give up their jobs when such individuals are responsible for the family incomes; in some cases, however, this procedure may be absolutely essential If the patient must continue at some kind of work for the support of his family, it may be possible

for the physician who does not wish to see many of his arthritic patients wandering off to quacks or to using patent remedies.[14] "He must even compromise at times with his intellectual honesty and employ methods of doubtful value to hold the patient to the pursuit of a few simple measures of proved value."[27]

In an effort to standardize just what is meant by "improvement" in rheumatoid arthritis, a special committee of the American Rheumatism Association, led by Dr Otto Steinbrocker, formulated criteria for grading improvement after a course of therapy. The criteria are listed below as they appeared in the report of this committee [30]

CRITERIA FOR EVALUATING THERAPEUTIC RESULTS IN RHEUMATOID ARTHRITIS[30]

(American Rheumatism Association)

To provide the most convenient and reliable means of assaying results, four categories of response of rheumatoid activity to treatment are recommended These range from Grades I to IV Grade I represents the maximum improvement, Grade IV, no response, and Grades II and III, intermediary stages The criteria are based entirely on OBJECTIVE EVIDENCE since *subjective symptoms are considered unreliable*

[illegible]

sis and fever periarticular swelling to have resolved

3 No new development of rheumatoid activity, including new joints, nodules, or tendonitis.
4. Remaining impairment of joint mobility not to be due to inflammation or muscular spasm.
5 The erythrocyte sedimentation rate within normal limits

The Committee recommends that a follow-up period be included in any therapeutic study to provide a comparison with the initial response to treatment in this condition If complete remission is maintained for at least five years, the disease may be considered arrested

GRADE II, MAJOR IMPROVEMENT

1 Absence of systemic signs of activity such as leukocytosis and fever
2 Signs of joint inflammation, such as heat, redness and tenderness, to have subsided completely
3 Minimal joint swelling, or articular enlargement due to irreversible changes or capsular thickening, may be present
4 No new rheumatoid process of intra-articular or extra-articular nature
5 Remaining impairment of joint mobility due to muscular spasm to be absent or minimal
6 Moderately elevated erythrocyte sedimentation rate may be present

GRADE III, MINOR IMPROVEMENT:

1 Any decrease in signs of activity inadequate for Grade II.
2. Increased functional capacity, general or local

In view of the natural course of the disease and the inherent difficulties in evaluating therapeutic agents over an arbitrary period of observation, the Committee believes that Grade III improvement should not be con-

to modify his job or his hours of work, or to provide additional rest periods during his work, or frequent short vacations.

Exercise in Rheumatoid Arthritis

1. Proper exercises are extremely important in treatment of the patient with rheumatoid arthritis *Too much exercise may cause some harm to the arthritic, but too little exercise may promote crippling.*
2. *At least one or two gentle movements of the affected joints* [illegible]

   within fatigue and pain limits.
3. **Never exercise a patient to the point of persisting pain or fatigue.**
4. Any motion which is attended, or followed within twenty-four hours, by an increase of pain, swelling or stiffness in the joints has been excessive; this must be reduced or modified. Proper exercises should cause no *severe* pain during their performance.
5. Even in the more chronic stages of an arthritis, not more than an hour *of continuous* exercises *should* be *performed Frequent short rests are* needed.
6. The occurrence of marked muscular spasm during an exercise is a signal for cessation of the movement.
7. Fibrillation of muscles indicates that the active exercise is too strenuous
8. All movements must be slow, deliberate and not jerky or tiresome.
9. All periods of strenuous exercise must be followed by an equal period of rest. See chapter on Physical Therapy for details.

Massage in Rheumatoid Arthritis

1. Unless contraindicated, every patient with non-acute rheumatoid arthritis deserves proper massage **Contraindications to massage** include an acutely inflamed joint, a temperature above 100° F, a suspicion of malignancy or phlebitis or increased pain, swelling or stiffness in a joint persisting (more than two hours) following the massage.
2. Begin the massage with gentle stroking and later use deeper stroking motions, kneading and friction of the *surrounding muscles for ten* minutes twice a day. This is best performed after at least fifteen minutes of heat to the tissues. See chapter on Physical Therapy for details

In patients with chronic rheumatoid arthritis, it is often advisable, in addition, to spend several weeks once or twice a year in a well-equipped hospital or spa for more intensive therapy, general check-up and possibly, the benefits of a vacation away from home. *The patient definitely must realize that 21 baths at some particular spa will not cure his rheumatoid arthritis.* The individual must be told that despite the claims of any particular spa concerning its mineral waters and their supposed virtue, *it has not been shown that appreciable amounts of any element are absorbed from such waters or muds,* but that the value of such spa or hospital therapy includes

sidered significant and, therefore, should not be included in any statistical summary of a therapeutic agent or procedure This is to avoid misleading lumping of favorable results.

GRADE IV, OR UNIMPROVEMENT.

1. Unaltered rheumatoid activity or unaffected functional capacity.
2. Exacerbation of any previously involved joint or joints, or development of new joint activity.
3. In the presence of the above, normal or lowered erythrocyte sedimentation rate not significant.

Subjective evidence by itself is regarded as unreliable and, without objective signs of response, patients are classified as unimproved.

BIBLIOGRAPHY

1 ADAMS, C H, and CECIL, R L Ann Int. Med, 33, 163, 1950
2 BALBONI, V G, HOLLANDER, J L, and KYDD, D. M.: Am. Jour Med Sci, 212, 153, 1946.
3 BARSI, I Brit Med Jour, 2, 252, 1947
4 BOLAND, E W, and HEADLEY, N E Jour. Am Med Assn, 148, 981, 1952
5 BOOTS, R H Unpublished Data
6 BOUCEK, R J, and LOWMAN, E W: Am Jour Med Sci, 215, 198, 1948
7 BROWN, T McP, WICHELHAUSEN, R H, MERCHANT, W. R, and ROBINSON, L B Am Jour Med Sci, 221, 618, 1951
8 COSS, J A., BAUMAN, E, BOOTS, R H, and LIPMAN, M O: Am Jour. Med, *4, 710, 1948.*
9 FORESTIER, J Jour Lab and Clin Med, 20, 827, 1929
10 FORESTIER, J, and CERTONCINY, A. Presse Med, 54, 884, 1946
11. FREYBERG, R H, *et al* Jour Clin Invest, 19, 423, 1940
12. GILLMAN, T, and GILLMAN, J Am Jour Med. Sci, 211, 448, 1946
13 GRANIRER, L W Jour Am Med Assn, 146, 915, 1951.
14 [illegible] P S, *et al* Ann Int Med 28 66 1948
15 [illegible] POLLEY, H. F. Proc. [illegible]
16. [illegible] s Co, Phila, Pa, 1952
17 [illegible]
18 HOLLANDER, J L, and HARRIS, T N Am Jour. Med Sci, 221, 342, 1951
19 HOLLANDER, J L, BROWN, E M, STONER, E K, and DEMOOR, P.: Jour Clin Invest, 30, 701, 1951
20 HOLLANDER, J L, BROWN, E. M, JESSAR, R A, and BROWN, C Y.: Jour Am Med Assn, 147, 1629, 1951
[illegible] and MANKLE, E A. Jour [illegible] 182, 1931. [illegible] A, VAILLANCOURT, [illegible] 93, 1952 [illegible] R M: Am Jour [illegible] 27 112 1942 [illegible] 40 [illegible]
29 SF[illegible] ELKIND, M.: [illegible] Am Med
30 ST[illegible] Assn, 140, 659, 1949
31 TALCOV, R H, and BAUER, W New England Jour. Med, 228, 152, 1943
32 TROMMER, P R, and COHEN, A Jour. Am Med Assn, *124, 1237, 1944*
33 TYSON, T L, and HOLMES, H.. "Rheumatic Diseases," W. B Saunders Co, Phila, Pa., p 131, 1952
34 ZUCKNER, J, KIDD, E C, BOYCE, K C, and FREYBERG, R H. Paper read before Am Rheumat Assn, Chicago, June 6, 1952.

contained sulfur. Preston and his co-workers[66] have clearly shown, however, that sulfur need not be contained in a gold compound to make it effective and, furthermore, that the salts identical with therapeutically effective gold compounds except that they contained no gold were ineffective to

in vitro Even though the organisms were grown in the presence of effective gold salts (*in vitro*), they did not lose their ability to produce arthritis Thus, it appears that the effectiveness of gold therapy with this type of animal arthritis is not just anti-bacterial or, if so, that it requires the host cell in which to function

Heilman[45] found that a single dose of gold sodium thiomalate injected

Angevine and Cecil[74] showed that gold compounds prevented hemolytic streptococcus arthritis in rats, but did not cure the arthritis once it was established

Animal investigations have been conducted[5] to learn in which tissues gold is

go

in

ubules show extensive degeneration and necrosis and the urine contains uch albumin In the convoluted tubules large aggregates of precipitated old or gold salts are found The pathologic changes are in proportion to e amount of gold administered, but it should be emphasized that in ese animal studies of toxicity the amount of gold injected was always ch greater than the amount used in treatment of humans The liver tains considerable gold but smaller amounts than are found in the eys.

en insoluble gold compounds in oil are injected intramuscularly, there ys a large amount of the material found at the site of injection and d content in the kidneys is less, as is the damage to the kidneys lloidal gold preparations are injected or ingested the liver becomes ith the particles which are quickly phagocytized by the reticulo- l cells, and the parenchymal cells are moderately damaged as

per cent of the instances of toxicity. Relapses were reported in one-fourth of all cases.

Controlled Evaluation of Therapeutic Effect of Gold Compounds.—In the English literature are reported three clinical evaluations of gold therapy in which simultaneous controlled observations were made. The first of these reports, that of Ellman, Lawrence and Thorold[23] is summarized in Table 9. All patients had active disease. Half of the patients who received

greater improvement, but also increased toxicity

TABLE 9

SUMMARY OF CONTROLLED STUDY OF GOLD THERAPY BY ELLMAN, LAWRENCE AND THOROLD

Patients	Gold Salt Used	Asymptomatic and E S R Returned to Normal	Some Improvement
		Per Cent	Per Cent
60	Gold Thioglucose	37	94*
30	Placebo sterile oil	3	76

* To greater degree than in controls

TABLE 10

SUMMARY OF THERAPEUTIC EFFECT OF THE CONTROLLED STUDY OF GOLD THERAPY BY FRAZER

Improvement	Patients			
	Treated with Gold	Treated with Placebo	Treated with Gold	Treated with Placebo
	Number	Number	Per Cent	Per Cent
Great	24	4	42 (56)*	8 (33)*
Moderate	12	6	21 (14)	13 (26)
Slight	11	11	19 (11)	24 (13)
None	5	10	9 (16)	22 (13)
Worse	5	15	9 (3)	33 (15)
Total	57	46		

* Figures in parentheses represent the patient's independent estimate of his progress

In 1945, Fraser[24] reported a study of 110 cases in which he controlled the effects of gold therapy All patients were suffering from active disease which had existed from two to five years Approximately half of the patients received injections of a placebo. Neither the patients nor the phy-

were observed for only one year. The results are summarized in Table 10 That the difference between the two groups indicates benefit from gold therapy cannot be doubted from these data. Frazer states that after treatment with gold, 17 of 19 formerly incapacitated patients were able to re-

LABORATORY INVESTIGATIONS OF GOLD THERAPY

It has been pointed out that chrysotherapy was added to the treatment for arthritis largely as a result of clinical trial. Much has been learned during the past decade regarding the effects of gold salts by research performed in the laboratory.

Gold Therapy in Experimentally Produced Arthritis in Animals.—It has been repeatedly shown[25,75,76] that when the proper technique is employed, polyarthritis can be consistently produced by the injection of pleuropneumonia-like organisms into mice or rats Careful study of the pathology and clinical course of this experimental arthritis shows that it is not the same disease as human rheumatoid arthritis. Many features of this experimental arthritis are similar to the human disease; the animals have fusiform swelling of proximal interphalangeal joints of toes, and of wrists, ankles, elbows, knees and sometimes, the shoulders or hips are affected Frequently the arthritis is symmetrical. Usually there is intense synovitis which initially is moderately purulent (a major dissimilarity to rheumatoid arthritis), usually the infecting organism is found in the joint exudate early

into it. This arthritis becomes chronic and progressive with newly affected joints becoming involved several weeks after the animal has been infected Ankylosis is common and deformities frequently result.

When this type of experimental arthritis was first described, there was much speculation as to whether pleuropneumonia-like organisms might be

tract in humans who do or do not have arthritis,[21] but to date they have not been isolated from the synovial fluid of arthritic joints except in cases of Reiter's syndrome.[22]) The similarity of this type of experimental arthritis to rheumatoid arthritis and the ease and dependability of its production afford excellent opportunity to evaluate methods of prevention and treatment which may have importance in therapy for human arthritis

Sabin and Warren[76] showed that this type of experimental arthritis could be completely cured when suitable gold compounds were injected early in the disease Colloidal gold, salts of bismuth, arsenic and antimony, sulfa drugs and sodium salicylate failed to cure. Some of these experiments were repeated by others[66] with similar results and it was shown further that the arthritis could be prevented by the same gold salts which were curative, if injected within a few days of infection. With insufficient doses of gold salt the cure was not complete or recurrences of illness spontaneously occurre

the radical to which gold was attached. Gold chloride was therapeuticau effective but quite toxic. The more effective gold salts with lower toxicit

TABLE 8

RESULTS IN 6,467 CASES OF RHEUMATOID ARTHRITIS TREATED WITH GOLD SALTS, REPORTED BETWEEN 1935 AND 1948.

Investigators	Year of Report	No of Cases	Period of Observation	Arrested or Greatly Improved	Moderately Improved	Slightly Improved	Duration of Improvement	Relapsed*	Toxicity**
			Years	Per Cent	Per Cent	Per Cent	Years	Per Cent	Per Cent
Forestier[28]	1935	550	6	75			2 to 3		
Hartfall, Garland and Goldie[42]	1937	690		66.7	13	6.2		20.9	40.0
Sashin, Spanbock and Kling[77]	1939	80	1 to 5	43.7	38.7			10.4	23.7
Goldie[38]	1939	400	½ to 4	76	10.5	4		18.5	17.0
Logefeil and Hoffman[54]	1941	139	1 to 5	56.8	24.5	14	1.8	13.7	12.9
Smyth and Freyberg[79]	1941	80		61			½ or more		33.0
Dawson, Boots and Tyson[20]	1941	100		51	25	9	½ to 2	12	50
Gardner[35]	1941	250		40	40			12	8.4
Cecil, Kammerer and de Prume[13]	1942	245	1 to 5	66	20		½ to 5	40	42
Price and Leichtentritt[67]	1943	101	1 to 3	60		12	1	55	38
Hartung[43]	1943	264		54				21	
Graham and Fletcher[40]	1943	95	1 to 3	67	20				51.5
Rawls, et al[71]	1944	100	1 to 4	53	21	12.			42.
Robinson[72]	1944	200	1 to 5	50.		25.			
Cohen, Goldman and Dubbs[15]	1945	259		64.		25.1			8.9
Oren[60]	1946	250		84.5			1 (Median)	7.2	4.6
Ragan and Tyson[64]	1946	142	3 to 6½	50	39.			75	30
Rose[73]	1947	91	½ to 1	73	11	5.5			20
Sundelin[84]	1948	2441	1 to 12	––––90––––***					50.
Total		6467						24.9	37.1

* Up to 80 per cent improved again on further therapy

** Includes mild toxicity not requiring cessation of therapy; only 4.5 per cent of reactions were severe

*** Degree of improvement not reported, simply "90 per cent were objectively and subjectively improved."

using equal doses of the compounds which differ greatly in their gold content.

lates as a gold-protein chemical complex. Neither has the chemical state of gold in urine and feces been established.

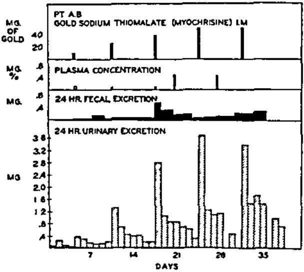


FIG. 81.—Plasma concentration and excretion of gold in a patient with rheumatoid arthritis treated with gold sodium thiomalate injected intramuscularly in increasing doses. (Courtesy of *Clinics*, J. B. Lippincott Company.)

When aqueous solutions of crystalline gold salts, organic or inorganic, are injected, there is a step-like increase in plasma gold content correspond-

re found in feces when larger amounts were injected. In the urine, larger ounts of gold were eliminated on the day of injection, and the amount rine increased in step-like fashion when larger amounts were injected uch phenomenon is exhibited in the fecal excretion of gold. The larger unt of gold found consistently in the urine on days of injection did not

Controversial Opinions.—Besides these controlled studies which definitely indicate benefit derived from gold therapy there are numerous statements which enthusiastically support this form of treatment In contrast are unfavorable reports of some very capable investigators. One of the earliest physicians[64] to report the use of gold salts in rheumatoid arthritis was "not favorably impressed" Snyder and Traeger[90] in their first (1939) report state that out of 50 patients treated only 1 showed "excellent" and 5, "good" improvement, in half of the cases results were "poor." However, later (1943) they reported[91] 48 per cent of patients previously "refractory" to commonly accepted forms of treatment had definite clinical improvement when treated with gold sodium thiosulfate. Cutts[17] states that the use of gold salts is attended with real danger and "even the best results are far from spectacular" Recently Browning *et al*,[9] using gold sodium thiomalate and gold acetyl cysteine, reported only 23 per cent of 47 patients were improved

There have been no more carefully studied cases than those of Short, Beckman and Bauer[78] who found little advantage in gold therapy determined by reviewing 35 reports of other investigators who employed gold salts when compared to their own results in 274 patients who received general and orthopedic treatment and no gold salts Their patients were observed one half to seven years Results (Table 12) show 16 4 per cent remissions and 52.9 per cent improvement without gold therapy Their review of literature showed only 22 6 per cent remissions and 59 3 per cent improvement in the patients treated with gold

TABLE 12

SUMMARY OF RESULTS OF GENERAL AND ORTHOPEDIC TREATMENT (NO GOLD THERAPY) FOR RHEUMATOID ARTHRITIS, SHORT, BECKMAN AND BAUER

State of Disease at End of Observation	Cases			
	Number		Per Cent	
Improved .	145		52 9	
In remission		45		16 4
Moderately		33		12 0
Slightly		67		24 5
No change	79		28 8	
Worse	50		18 3	
Total	274			

Nature of Therapeutic Effect of Gold Salts—Statistics showing percentage improvement are not the only basis for judging the value of gold therapy. The nature of the improvement and the speed with which it occurs are also important. Many investigators have shown that not in-

shown by cloudy swelling and edema The kidneys contain very little gol and show no significant histologic changes. No gold salt is seen precip tated in the tubular cells.

The gold retained in the animals is not found concentrated in any par ticular somatic tissue but is distributed through all tissue roughly in propor tion to the amount of cells therein. Gold is not concentrated in synovia fluid.

STUDIES OF THE METABOLISM OF GOLD IN HUMANS

Many investigations of the absorption, circulation and excretion of gol have been conducted over long periods of time during treatment of patient with different gold preparations. Such studies could be conducted onl after the development of a highly sensitive, selective and accurate metho for quantitative analysis for gold in biologic fluids and tissue.[34] In thes metabolism studies gold compounds which are commonly used in therapy and which are representative of preparations which have different chemica and physical characteristics, were employed (Table 7). Preparations o

TABLE 7

PREPARATIONS REPRESENTING DIFFERENT CHEMICAL AND PHYSICAL TYPES OF GOL COMPOUNDS

Gold compound	Proprietary name	Solubility in water	Physical state	Gold content of compound per cent
Gold Sodium Thiosulfate	Sanochrysine Crisalbine Sanocrysin	Yes	Aqueous solution	37
Gold Sodium Thiomalate	Myochrysine	Yes	Aqueous solution	50
Gold Thioglucose	Solganol-B-Oleosum	Yes	Oil suspension	50
Gold Calcium Thiomalate	Calcium Aurothiomalate	No	Oil suspension	50
Gold Thioglucoanalide	Lauron	No	Oil suspension	54
Gold Sulfide	Aurolsulfide	No	Aqueous colloidal suspension	87

inorganic and organic salts, aqueous soluble and insoluble compounds, crystalline solutions and suspensions in oil have been studied; their chemical and physical differences should be expected to account for different metabolic and therapeutic behavior. Since the effects of all gold compounds depend upon the amount *of gold* they contain, to compare different preparations it is necessary to employ doses of the different preparations which provide equivalent amounts of *gold*; comparison cannot be made by

sume employment; of the control, only 4 of 14 could return to work. There was a more marked fall in the erythrocyte sedimentation rate in the

logical benefit and spontaneous improvement due to natural causes. Treatment was stopped in four of the gold treated group and three of the control group because of "toxicity" of the injected material. (The "toxicity" considered to exist in patients who received no gold will receive further comment below). Frazer prudently points out that the better results claimed for gold therapy by the majority of other physicians are unjustifiably high because of the lack of adequate control observations (a criticism which applies to practically all evaluations of separate forms of treatment for rheumatoid arthritis).

TABLE 11

SUMMARY OF THERAPEUTIC RESULTS IN THE CONTROLLED STUDY OF GOLD THERAPY BY WAINE, BAKER AND MATTIER.

State of Disease at end of Observation	Treated with Gold	Treated without Gold	Treated with Gold	Treated without Gold
	Number	Number	Per Cent	Per Cent
Arrested	12	6	20 7	9 7
Markedly improved	21	12	36 2	19 4
Improved	14	17	24 1	27 4
Unimproved	11	27	19	43
Total	58	62		

The most recent carefully controlled study of chrysotherapy in rheumatoid arthritis is that of Waine, Baker and Mettier[83] who studied 120 patients with active rheumatoid arthritis All patients showed characteristic constitutional features of this chronic disease as well as symmetrical polyarthritis and an elevated erythrocyte sedimentation rate. All patients were seen by members of an arthritis committee one to four times monthly during the

average duration of treatment and observation was 3.02 years. Injections of gold salts (gold sodium thiosulfate and gold sodium thiomalate) were ... new patients and ... reatment with- ... efore admission was 5 5 years and the average period of treatment and observation was 2 9 years. All of these patients were given general care similar to that used in the control group Gold salts were injected without rest periods in the ... bi- ... al ... ts

apparently arise from higher plasma concentrations for between injections, content of gold in the plasma remained relatively constant The rather sharp decrease in urinary excretion of gold after the day of injection suggests that soon after injection, gold is absorbed into the blood and circulates through the kidneys in a state that will be eliminated readily in the urine, and that after a longer time less gold remains circulating in this readily excretable form.

urine and 2 mgm in the feces, so that if one disregards the gold injected previously and considers only that injected on the eighteenth day, it is found that 76 per cent was retained in the body between the time of two injections. With continued injections this retained gold accumulates more and more Since the excretion of gold does not increase significantly after

g treat-

uring a

actual

measurement in two patients to be 81 and 86 per cent of the amount injected

After treatment is stopped, gold is found in the blood and is excreted for long periods of time, the duration depends chiefly on the size of the weekly dose because that determines the amount of retention of gold during the treatment period In patients who receive weekly injections of 50 mgm of *gold*, gold continues to circulate and be eliminated in decreasing amounts for many months (observed up to fifteen months). With weekly doses of 25

two or three months after cessation of treatment

higher blood concentration for a few hours after injection. The retention

sorbed slightly more slowly and produces more variable and slightly lower (about 70 per cent) concentrations of gold in plasma than obtained with solutions of gold salts. Excretion is similar to that resulting from injections of

most likely because there are greater variations in absorption from the

17

In late stage irred the only value gold therapy can be is to check the progress of the disease and thus

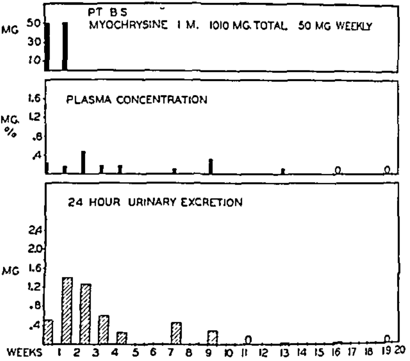
practice in some clinics. If this is the basis for the use of gold therapy, results will of course not be impressive because of the joint damages already present Such is, no doubt, the basis for poor results experienced by some physicians who studied this form of treatment in the earlier days of gold

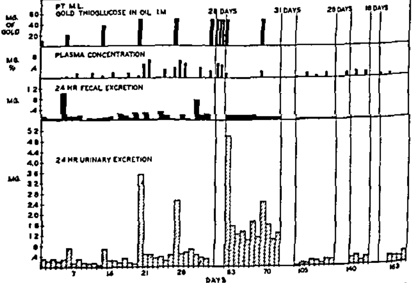
tying.

No hard and fast rules can be laid down for the institution of gold therapy. Because of the potential toxicity of gold, the decision to employ this treatment in any patient should be made only after careful considerations of all circumstances and with seasoned judgement *It is unwise to*

months Furthermore many cases are not severe, indeed some are mild, and a good program of treatment without the use of gold salts may be re-

to be advancing, to involve articulations not previously affected and causing increased damage to joints already involved, at that time, it seems reasonable and correct to consider seriously one of the special forms of supplemental treatment, including gold salts (See also discussion relating to selection of cases for cortisone or corticotropin in Chapter 17) Gold therapy should not be used unless the physician and the patient know what can be expected of it therapeutically, and appreciate its toxic potentialities When, without its use, the arthritis is progressing, and one knows of the serious consequences of the continuing active disease certainly there is wisdom in using a form of treatment that may arrest or significantly decrease the activity of the process, even though the treatment has potential toxicity As "last resort" therapy, gold salts are often used too late to obtain best results Decision concerning the use of this form of therapy


PT B S
MYOCHRYSINE I M. 1010 MG TOTAL 50 MG WEEKLY
MG
50
30
10
PLASMA CONCENTRATION
MG %
1.6
1.2
.8
.4
24 HOUR URINARY EXCRETION
MG
2.4
2.0
1.6
1.2
.8
.4
WEEKS 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20

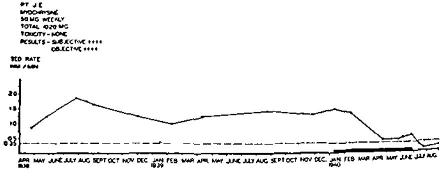

PT M L
GOLD THIOGLUCOSE IN OIL I M
MG OF GOLD
28 DAYS
31 DAYS
29 DAYS
18 DAYS
PLASMA CONCENTRATION
24 HR FECAL EXCRETION
24 HR URINARY EXCRETION
DAYS

dramatic in comparison with the speed of effects of cortisone and corticotropin, nevertheless it is important and no statistical report on gold therapy takes into account this important factor. When a result such as por-

shown in this graph was incapacitated from rheumatoid arthritis for two years during which different forms of treatment were employed with little or no benefit. The change in sedimentation rate was paralleled by clinical *improvement until complete arrest of the disease state occurred* and the patient returned to work.

Considering this complex controversial question from all aspects and

ponents of gold therapy are assisting greatly in preventing unwarranted enthusiasm for this treatment which indeed has definite limitations (to be discussed later) based chiefly upon its potential toxicity, undependable effects, and the high relapse rate of disease activity after gold therapy has been discontinued.



Company)

Selection of Cases for Treatment with Gold Salts.—The selection of the patient suitable for gold therapy presents another controversial question This is of course an exceedingly important consideration and one about which there should be little or no debate if decision is based upon a knowledge of (1) metabolism and pharmacology of gold, and (2) the nature of the disease. In this connection it should be emphasized that *gold can be helpful to patients with rheumatoid arthritis in only one way, that is by helping to arrest activity of the disease.* This is the only therapeutic effect that gold can contribute; *it does not repair damaged cartilages or bones, nor does it restore deformed and ankylosed joints* Therefore, benefit from gold salts can be expected only when employed during the *active stage of the disease*

Only small amounts of gold are found in feces and usually *none* in the urine Usually less than 1 per cent of the gold injected can be found in excreta, hence leaving 99 per cent retained A study of the tissues of rats treated

are measured one, two, four, six and twenty-four hours after intravenous

hours However, the gold in the urine decreased after two hours As early as thirty minutes after intravenous injection of colloidal gold sulfide, no gold was found in the plasma, and none was found in the urine (it was

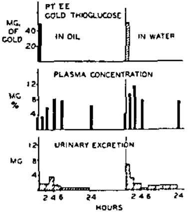


Fig. 85 — Comparison of absorption and excretion of gold for twenty-four hours following the injection of gold thioglucose solution and suspended in oil (Courtesy of *Clinics*, J B Lippincott Company)

phagocytosed). All aqueous preparations injected intramuscularly behave similarly Quick absorption from muscle depots is noted Practically no gold is absorbed from intramuscular depots of the insoluble gold calcium thiomalate during twenty four hours after injection This is in sharp contra ... A somewhat ... fter intramuscu... suspended in oil Here again the values are about 70 per cent of those obtained with equivalent amounts of solutions of gold salts Figure 85 shows that gold thioglucose behaves about the same if injected in aqueous solution or suspended in oil

Mode of Action of Gold Salts Used in the Treatment of Rheumatoid Arthritis.—The way in which gold salts act to cause their effects when used in the treatment of arthritis is not known Much study has been conducted to attempt to learn this, but with unsuccessful results It has been shown[n]

develop on the palms and soles and soon be followed by generalized exfoliative dermatitis. Discrete lesions resembling lichen planus may appear localized to a few parts of the body or may be quite generalized. Such an eruption usually persists several months and heals slowly. Lesions re-

arms, legs and trunk, sometimes, purpura may not be due to thrombocytopenia, but apparently to endothelial damages. Pre-existing tinea, or seborrheic dermatitis, frequently becomes aggravated during the course of treat-

of gold toxicity, any eruption, especially if it is pruritic, which develops during gold therapy must be considered with suspicion until its nature becomes clarified. Gold dermatitis usually becomes progressively worse if

In two patients, the author has observed complete alopecia to persist for three years.

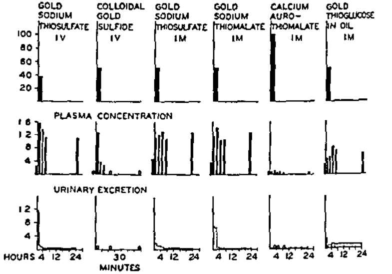
Mouth Lesions.—Oftentimes, along with dermatitis, stomatitis occurs, it may appear without skin eruption A "metallic taste" may precede stomatitis. Discrete inflammatory lesions develop in the oral mucous membranes; these usually ulcerate, so that ulcerative stomatitis, glossitis or gingivitis results. Like gold dermatitis, mucous membrane lesions may be

Nephritis.—Toxic nephritis may be caused by gold. Slight transient albuminuria may occur during gold therapy, and have no relationship to this

muscle depots where the oily mass is injected in contrast to the quite uniform behavior observed in different patients treated with solutions of gold salts.

A distinctly different picture is observed when the insoluble salt, gold calcium thiomalate in oil, is injected intramuscularly. The plasma gold concentration is very small in most patients; there is much variation from time to time in the same patient during the period a constant weekly dose is injected, and the plasma gold level varies considerably from patient to

when solutions of gold salts are injected. There is greater variation from day to day, and on many days no gold is found in the urine. These findings clearly indicate that absorption of this insoluble gold salt in oil is sharply



... content of one two four six

retarded, consequently, there is greater retention than when soluble gold

... onable to assume

ifferently in the
fter intramuscu-
found in more

than traces in the plasma. Even when injected intravenously, little or no gold can be found in the plasma as soon as thirty minutes after injection

will be just as difficult if postponed; for best therapeutic results, an early decision is reasonable. If, on first consideration of gold therapy, it is decided against, the question should
months unless the disease is abating.
the use of gold therapy need be mac
after carefully evaluating the potent
the risks of the toxicity of the drug.

for treatment with gold salts should be on the same basis as for adults Most investigators also consider that in patients with psoriasis who have rheumatoid arthritis the problem is the same as in the person with uncomplicated rheumatoid arthritis. (Psoriasis is not a contraindication to the use of gold) In the United States, ankylosing arthritis of the spine (Marie-Strumpell disease) is usually felt to be rheumatoid arthritis of the spine—rheumatoid spondylitis Results of treatment of this disease with gold salts have for the most part been very unsatisfactory. Moreover,

rheumatoid spondylitis.

TOXICITY OF GOLD

The greatest problem in connection with treatment with gold salts is presented by the potential toxicity of gold. This is also the chief reason that gold therapy is not more generally employed. The toxic potentialities of gold justify hesitancy in selecting this form of treatment.

In humans the toxic manifestations of gold may affect many different organs and systems. Cutaneous manifestations are by far the most common.

Gold Dermatitis.—With few exceptions the eruption is preceded by

neck and thighs after the first two or three injections of small amounts of gold compound. This type of dermatitis, usually disappears within a few days, is not severe and often gold salts may subsequently be tolerated well.

at the neck, axillae, groin, intercrural regions, the chest and back, where the lesions are discrete, oval and rosaceous, resembling pityriasis rosea The lesions have a thin wrinkled epidermal surface. This form of rash usually persists many weeks, and is very pruritic. The lesions may slowly

the controversy regarding gold therapy.

CLINICAL EXPERIENCES WITH GOLD SALT THERAPY

Among the many difficult clinical problems in modern medicine none is harder than the evaluation of some forms of treatment for rheumatoid arthritis At the top of this list is the problem of assaying gold therapy It is of course reasonable to expect differences of opinion and uncertainty when one appreciates the many factors which need be considered Concerning the illness—the cause for the disease is unknown, the pathogenesis of the characteristic inflammatory changes is not understood, the disease is one of protean nature and one which travels an unpredictable course in every patient, it is usually characterized by remissions and relapses. Concerning the treatment—the gold compounds employed are numerous, they vary greatly in chemical and physical nature, the manner of treatment and dosage have been decidedly different, there is uncertainty regarding the manner of action of gold, the type of illness and stage in which treatment is employed differ in the experiences of many physicians, the underlying honesty or prejudice influencing the evaluation and conclusions, all are important factors To these must be added the lack of uniform criteria for classifying the status of the disease, and what constitutes significant change,[42] and the difficulty in allocating the true portion of change attribu-

Didactic statements cannot therefore be confidently made regarding the proper place of gold therapy in the program of treatment for rheumatoid arthritis Those who have had experience with this form of treatment or who have made special investigations concerning it usually have a firm conviction regarding this important question It should be instructive, therefore, to review the clinical reports concerning gold therapy

Many of the experiences reported prior to 1949 are summarized in Table 8 Not included in this table are three controlled studies which will be discussed separately This tabulation is presented, not for the purpose of allowing summation of experience which cannot be accurately done (see above) but to show the trend of the reports and for ready reference The shortcomings in the data and the lack of controlled observations are obvious. Nevertheless, it should be noted that among the nineteen reports of results in more than 6,000 cases, the majority of patients were considered to be significantly improved as the result of gold therapy In approximately one-third of all cases manifestations considered to be indicative of gold intoxication are listed, although these were of a severe nature in only 4.5

as shown by observations of Fraser[26] who found that 19 (27 per cent) of 49 patients among the control group *who received no gold* had what was considered *by the physician* as manifestations of "toxicity." These disturbances he listed as affecting the skin in 16 cases, the kidneys in 2, and the mouth in 1 case. If a comparable number of the patients in his series who were treated with gold salts had such "false toxicity" the incidence of intoxication *would be reduced to 38 per cent, a figure almost identical with the average* noted above.

Toxicity from gold may occur *at any time* following the first injection of gold salt, throughout the course of treatment and for several months after treatment has been discontinued. Usually, it appears after 300 to 500 mgm of the gold salt have been injected. Toxicity is usually mild, but approximately 5 per cent of the toxic reactions represent severe illnesses often very



FIG. 87.—Microscopic section of rat kidney stained with hematoxylin and eosin showing the extensive damage produced experimentally by cellular toxicity due to gold when gold sodium thiosulfate is injected in *large* doses.

prolonged and uncomfortable. Deaths have occurred from gold intoxication usually *from thrombocytopenic purpura, aplastic anemia, granulopenia* or ulcerative enteritis, very rarely from severe (complicated) exfoliative dermatitis. Mortality was as high as 3 per cent in 1935, between 0.5 and 0.6 per cent from 1935 to 1939, and recently approximately 0.4 per cent. The reduced mortality is attributable to (1) employment of smaller individual doses of gold salts, (2) improvement in some gold compounds, (3) greater knowledge of metabolism of gold and clinical response, (4) greater precautions during treatment and, (5) better treatment of toxicity (very recently).

Pathogenesis of Gold Toxicity.—There has been much research aimed at clarifying the nature of gold toxicity but with quite discouraging results

that gold salts have a marked chemotherapeutic effect against hemolytic

hemolytic streptococci. Hartung and Cotter[41] found serum of patients

microbe

shown that some inorganic gold salts inhibit oxygen uptake by liver and kidney cells, and that some organic non-ionizable gold compounds did not. Significance of these observations cannot yet be evaluated in relation to action of gold in the treatment of rheumatoid arthritis. That effects of gold are on host cells is supported by the observation of Sabin,[75] that gold salts known to prevent or cure arthritis caused by *pleuropneumonia-like* organisms in mice did not inhibit the growth of these microbes *in vitro.*

Recent animal studies of Libenson[54] tend to indicate that gold may operate in the tissues to compete for the SH radical in such a way as to alter the cellular metabolism in a manner favorable to the patient with active rheumatoid arthritis.

Because many gold compounds used in treatment and in animal experi-

gold salts

not depend upon the concentration of the element in or about the [illegible] joints. Perhaps it acts to stimulate the adrenal cortex.

Evidence (to be discussed more fully below) clearly indicates that the only benefit gold salts may have in treatment of rheumatoid *arthritis* is to

treatment. But frequently albuminuria is due to toxicity from gold and indicates existence of toxic nephritis. This is usually tubular degeneration but some glomerular lesions occur and hematuria is sometimes a feature of this complication. The nephritis may be trivial and short in duration, or quite severe and pass through a subacute phase with elevated blood pressure, retinitis and systemic effects before it subsides, months after onset. This form of toxicity has not been sufficiently appreciated or described

Hematologic Disorders.—Blood dyscrasia, as a form of gold toxicity rarely occurs, but when it does it may be serious or even fatal. Thrombocytopenic purpura,[20,78] hypoplastic or aplastic anemia, granulopenia and secondary anemia have been reported. Occasional fatalities have been ascribed to subdural or cerebral hemorrhage caused by hemorrhagic purpura.[87] Eosinophilia frequently precedes or accompanies gold toxicity, especially dermatitis, hence its presence suggests impending or existing toxicity but, frequently, esoinophilia may develop in arthritic patients who are receiving no special treatments, so it cannot be relied upon as a dependable diagnostic aid.

er.

m

Such symptoms

hich

tis"

sual

preparations of gold salts have been reported to be as high as 50 per cent (Table 8) and as low as 4 per cent.[61] In one series[83] of 30 cases treated with gold thioglucoanalide, no reactions occurred. The average incidence of all reactions in a large group of cases was 37.1 per cent (Table 8). It should be emphasized that these figures of incidence include all minor reactions,

sc ... of treatment, indeed

sc ... [20]

C ... nd

to be only 4.5 per cent.

In one series of 100 carefully evaluated cases,[20] toxicity was listed as follows: pruritus—50 patients; dermatitis—48; stomatitis—25; gastro-intestinal symptoms—19; hematologic changes—19, renal disease—2; hepatitis—2 Five patients developed exfoliative dermatitis and 3, severe stomatitis. *One patient died of aplastic anemia, after she had had radium*

granulopenia—1. *It should be emphasized*, however, that in these series cited, larger doses of gold salt were used than those which are now currently advised.

during gold therapy as manifestations of gold toxicity.

as shown by observations of Fraser[28] who found that 19 (27 per cent) of 49

would be reduced to 38 per cent, a figure almost identical with the average noted above.

Toxicity from gold may occur *at any time* following the first injection of gold salt, throughout the course of treatment and for several months after treatment has been discontinued Usually, it appears after 300 to 500 mgm of the gold salt have been injected Toxicity is usually mild, but approximately 5 per cent of the toxic reactions represent severe illnesses often very

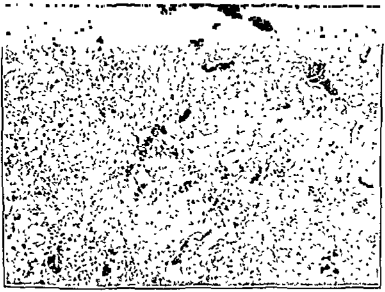


Fig 87 —Microscopic section of rat kidney stained with hematoxylin and eosin showing the extensive damage produced experimentally by cellular toxicity due to gold when gold sodium thiosulfate is injected in *large* doses

dermatitis Mortality was as high as 3 per cent in 1935, between 0 5 and 0 6 per cent from 1935 to 1939, and recently approximately 0 4 per cent The reduced mortality is attributable to (1) employment of smaller indi-

recently).

Pathogenesis of Gold Toxicity.—There has been much research aimed at clarifying the nature of gold toxicity but with quite discouraging results

A few preparations of gold not commercially available have been studied *Gold calcium thiomalate*, first prepared and studied by Sabin, has been tried clinically. This is an insoluble salt containing 50 per cent gold, suspended in oil for intramuscular injection *Sodium succinimido-aurate* is a soluble gold salt which has been prepared for intramuscular injection

To select the best preparation from those available, one should keep in mind the knowledge provided by metabolism studies Colloidal preparations seemed attractive because they provoked practically no untoward reactions. However, it was clearly shown that the colloidal particles are [illegible] that no pharma- [illegible] a fact both by [illegible] compounds *For* [illegible] *not recommended for treatment*

[illegible] that the insoluble [illegible] ylactic and thera- [illegible] uropneumonia-like organisms, and that it has a toxicity ratio in mice lower than other (soluble) salts Hence, trials of this salt in humans were made with great hope that this would be a better compound for treatment of rheumatoid arthritis However, results have been very unpredictable, chiefly because absorption varies so greatly. Furthermore, since toxicity in humans is apparently due to different factors than prevail in experimental animals, toxic manifestations were frequently observed with the use of this salt in patients Toxic reactions were often quite severe and very prolonged, most likely because of the large reservoir of gold salt at the site of injection (see page 258) The feature of insolubility which prompted development of this salt, with the hope that it would be less toxic, appears to be definitely a disadvantage [illegible]

[illegible]

this insoluble gold salt. In our present knowledge it seems quite clear that all insoluble salts have less advantage than disadvantage as therapeutic agents in humans. It is more reasonable to expect improvements in gold compounds to come from the development of preparations which will be more readily and uniformly absorbed, circulated, and excreted rather than substances that are insoluble and that are absorbed and eliminated so very slowly.

For treatment of humans, therefore, it seems at this time wise to select a soluble gold salt to be injected intramuscularly Because of the ready ab-

of large amounts of gold salts providing very much more gold than

portional to the amount of gold injected. Death occurs regularly if an amount of gold exceeding a certain minimal lethal dose is administered

clinical forms of toxicity have not been observed.[33]

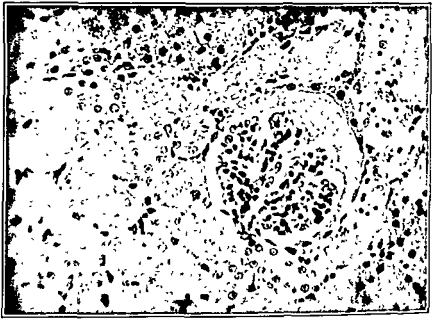


FIG 88 —Higher power magnification of a section of the same kidney shown in Figure 96. Note the marked cloudy swelling and necrosis of tubules, the glomerular congestion, the albuminous urine and the deposits of gold pigment in convoluted tubules

Skin from patients with gold dermatitis contains no greater concentration of gold than skin from patients receiving comparable amounts of gold, who have no toxicity,[33,34] showing that toxic dermatitis does not result [illegible] This form

[illegible]chymatous
[illegible]he cause for
[illegible]ploys large
doses of gold salt given at frequent intervals. [illegible] indications
that gold toxicity in humans is an allergic type of reaction: the occasional

sorption of s[illegible]
salts, there
injections.
appears to be the type of gold preparation to be preferred The soluble salts most commonly used in treating humans are gold sodium thiosulfate, gold sodium thiomalate and gold thioglucose There is little difference in the effects of these s[illegible]
be slightly more tox
gold and sodium.
sodium thiosulfate.
gold sodium thiomalate and gold thioglucose but some investigators[10,75] have thought that the latter is slightly less toxic. This may be more ap-[illegible]

in effective dosage of the salts There is certainly little difference between gold sodium thiomalate and gold thioglucose in oil; both are popular in this country.

Dosage Schedule.—The method of administration of gold salts has varied considerably among recent investigators. Because smaller doses have given results as good as larger doses which were customary, and because toxicity is less frequent and less severe with smaller weekly doses of gold salts the following schedule has been adopted by the author, and many other investigators

Of a soluble salt containing 50 per cent gold (gold thioglucose in oil, or gold sodium thiomalate) injections are given intragluteally at weekly intervals. For adults of average size:

1st injection	10 mgm of the salt
2nd injection	25 mgm. of the salt
3rd injection	50 mgm. of the salt
4th to 16th or 20th injection	50 mgm of the salt

If the patient has shown no improvement after this amount of treatment, it [illegible]

treatment less energetically for a prolonged period in an effort to reduce [illegible] incidence of relapse (seen to be so high). Injections of 50 mgm are then spaced at two week intervals and continued for 6 to 10 more injections after which this dose is given at three week intervals for several months and finally at monthly intervals for six to twelve months. The time to discontinue gold therapy cannot be arbitrarily determined; this depends upon the course of each patient. Observation during such a schedule of treatment strongly suggests that this "maintenance" treatment does help to reduce the relapse rate. In the event that a relapse occurs during this schedule when injections are given at two, three or four week intervals, resumption of weekly injections is advised, to continue for 6 to 16 more in

occurrence of dermatitis or blood dyscrasia after only three or four injections of gold salts; the development of toxicity following a *single* small

; following completion
hout intolerance, the
during toxicity, these
With this tentative
e of this mechanism

Patch and intradermal skin tests with gold compounds, used in therapy, showed no more positive reactions in patients having gold toxicity than in those who did not. Neither did such tests forecast future toxicity. These observations suggest that elemental gold and the compounds used for treatment do not themselves act as allergenic agents to cause gold toxicity.

Because gold is transported in the protein fraction of the blood plasma and because the urinary excretion of gold falls off sharply a few hours after

the nature of gold toxicity.

TABLE 13

THE INCIDENCE OF TOXICITY IN 126 PATIENTS TREATED WITH DIFFERENT WEEKLY DOSES OF GOLD SODIUM THIOMALATE (FREYBERG[19])

Weekly Dose of Gold Salt	Patients Treated	Incidence of Toxicity (All Forms)	
Mgm	Number	Number	Per Cent
100	46	19	41
50	63	19	30
25	17	3	18

Prevention of Gold Toxicity.—*All gold salts that may have therapeutic value have toxic potentialities!* Unfortunately there is no reliable method of predicting or testing to determine which persons will develop

showing us that the smallest amount of gold that may give satisfactory therapeutic results should be employed, for if toxicity develops it may be

m
be
th
pl

is

jections, the number depending upon the progress of the patient. Thereafter, an increase in the interval between injections is again made, as was formerly done.

The dosage schedule for children is similar except that the amount of gold salt used for such injection is reduced in proportion to the body weight or surface area

It is necessary to follow the precautions against toxicity already discussed (page 273). Should toxic manifestations be encountered—and they [illegible]

gold therapy is resumed following cessation of previous treatment.

Boots[8] reported that, with a weekly dose of 50 mgm of gold thioglucose in oil or gold sodium thiomalate, toxicity was less but the therapeutic results were not as good as when 100 mgm of the salt were injected at weekly intervals Rawls[70] reported good results and less toxicity from 25 mgm of gold salt injected at weekly intervals He usually began treatment with 5 mgm. of the salt injected intramuscularly twice weekly for three weeks then gave 25 mgm weekly. If no improvement occurred with this dose he in- [illegible]

[illegible] dosage schedules have been recommended by different physicians but these appear to have no advantages over the schedule outlined above

Contraindications to Gold Therapy.—The chief contraindication to the use of gold salts is previous, severe toxicity produced by gold or other heavy metals. Many other contraindications have been reported but experience has demonstrated that many conditions formerly thought to prevent the use of gold actually do not Allergic diseases have been considered by some physicians a contraindication, but it has been shown frequently that patients with asthma, hay fever or other allergic diseases can tolerate gold therapy quite satisfactorily If gold therapy is used in persons who have [illegible]

rheumatoid arthritis are benefited by pregnancy to such an extent that gold therapy is seldom desired It is generally considered that gold salts [illegible]

tution of gold therapy which is always more easily managed when complicating illnesses do not exist Previous kidney or liver disease is not a contraindication to gold therapy, existing diseases of these organs contraindicate only if they seriously impair the functions of these organs Even then, the danger is not that toxicity is more apt to occur, but rather that if severe

nificantly (Table 13). But *more important even, is the fact that the severity* of reactions was likewise usually reduced when smaller weekly doses were employed There is, therefore, a quantitative (but not absolutely proportional) relationship between the size of the weekly dose and the incidence and severity of toxicity of gold, which argues for the use of smaller doses.

During the early studies of gold metabolism it was hoped that a plasma gold level or an excretion rate of gold would be found to indicate the amount of gold required to produce benefit and the amount which would be likely or certain to produce toxicity (as is practical with potassium thiocyanate or sulfonamide therapy). *Unfortunately, such is not the case; clinical gold toxicity is not related to plasma gold concentration or to rate of excretion of*

Much effort has been expended in an attempt to improve the gold com-

which have been far less toxic, have had little or no therapeutic value Many insoluble crystalline preparations have been developed (including gold calcium thiomalate and gold thioglucoanalide) but undependable results are obtained with all insoluble gold compounds (as noted above), and although the incidence of toxicity is reported[72,83] to be less than with other gold salts, instances of severe exfoliative dermatitis and ulcerative lesions of oral mucous membranes, with recurrences lasting more than two and one half years, have been observed.[30] These recurrences suggest that from time to time there are auto-inoculations from the large reservoir of gold salt retained at the sites of injection when *insoluble preparations are employed* For these reasons *insoluble gold salts are not recommended by the author for treatment* They may possess the advantage that lethal toxicity may be obviated for as yet none has been reported. Instances of prolonged mild ... have been observed, however.[30] That large doses of insoluble gold

for, of

, prob-

which

... can be assured

those physicians who

ith the manifestations

treatment. The phy-

ach injection to inter-

... concerning the occurrence of *pruritus, skin eruption, purpura, sore*

r unusual symptoms *Urinalysis for*

performed weekly until 500 mgm. of

toxicity from gold is encountered, the summation of difficulties may produce serious consequences Similar considerations apply to uncontrolled diabetes, severe blood dyscrasias, including purpura, hemophilia and severe anemia. Granulopenia usually is caused by a drug sensitivity which should

organs is good and if the patient is in good state of nutrition and general health. Debilitation from any cause should militate against gold salt therapy.

Disseminated lupus erythematosus is frequently made worse by gold therapy and the disease is not benefited by gold even if the drug is tolerated, hence this and other diffuse collagen ("vascular") diseases are virtually a contraindication to this form of treatment.

Mild or moderate anemia is not a contraindication to treatment with gold salts Anemia is very common in patients with rheumatoid arthritis, and, frequently, it improves when proper antianemic treatment is employed during gold therapy.

Precautions During Gold Therapy.—The clinical and laboratory observa-

ment is employed in any patient the physician should frankly discuss the nature of treatment with the patient who should know the potentialities for toxicity as well as therapeutic benefit. Only with the patient's knowledge and consent should gold therapy be employed. Some physicians and clinics demand each patient to sign a statement releasing the physician from responsibility if toxicity results. The chief advantage of such a "release" seems to be the requirement of a full explanation of all aspects of gold therapy to the patient before such treatment is begun

Simultaneous Use of Gold Salts and Cortisone or Corticotropin.—It has been hoped that advantage would accrue from the use of gold salts during and following the administration of cortisone or corticotropin. Many patients have been treated with cortisone until the rheumatoid arthritis became suppressed, then gold salts were begun with the hope that this form of treatment would maintain the benefits of the hormone which could then be discontinued and thus avoid the complications of prolonged hormone therapy. However, results have been disappointing; relapses follow cessation of cortisone in a way exactly as they do when no gold salts are given. The possibility that the slow action of gold salts might account for the failures, prompted a trial of initiating treatment with gold salts and then beginning the use of cortisone six or eight weeks later Several investigators[47] have reported that this has little or no value, indeed

latelet count. Leukopenia below 4000, quick reduction of hemoglobin,
rease in platelet count to below 75,000 per c.mm , albuminuria, hema-
ia, pruritus and stomatitis are all danger signs, though they may not be
stant or reliable signs of toxicity. *Occasionally, the erythrocyte sedi-*

some physicians to be prophylactic against toxicity, have been found by
st investigators to be of little or no benefit

Treatment of Gold Toxicity.—If gold toxicity has developed, further
ninistration of gold salt should be discontinued, symptomatic and sup-
tive care should be instituted and appropriate corrective treatment
mptly employed. Mild cases of erythema and pruritus may disappear
a few days and are relieved by calomine lotion with 0 5 or 1 per cent
enol Sedatives in small doses are helpful Treatment of generalized
matitis, especially exfoliative dermatitis, is best done in a hospital
enol-containing lotions and ointments and colloidal baths help to relieve
hing. In places where pruritic dermatitis persists, small amounts of

openic purpura and, when done, may be followed by prompt, excellent
provement Penicillin is advised during granulopenia for prophylaxis
inst infection

The Use of BAL —Many attempts have been made to alter the metabol-

toxic effects
tiveness again
t was logical t

Batterman, Traeger and Wainerdi[7] have reported that the benefits of each form of treatment—hormone and gold salts—were lost if the two were [illegible]

[illegible]

arger percentage than observed by other investigators to continue in [illegible] not believe there [illegible] dered gold quite [illegible] sed the question as to whether the problem is different when corticotropin is used instead of cortisone during gold salt therapy. Further research is needed to answer many questions concerning the simultaneous use of these hormones and gold, currently most investigators consider that such treatment is ineffectual or impractical.

BENEFITS AND LIMITATIONS OF GOLD SALTS

When gold salts are beneficial, the improvement is usually very slow. The patient should be forewarned that several weeks may pass before he notes any benefit, and that many months of treatment may be required before there is improvement sufficient to decreased disability. When the therapy is beneficial, signs of inflammation gradually decrease, pain lessens and function improves. However, the physician should remember that this form of treatment does not help all patients and that no test exists to indicate whether or not it will be beneficial except by clinical trial. The [illegible] gold salts, just as [illegible] development of [illegible] in favor of the patient.

As is true with all other forms of therapy, gold salts are not complete [illegible]

[illegible] palindromic rheumatism).[?] Gold salts cannot benefit patients with osteoarthritis, rheumatic fever, gouty arthritis, and other forms of rheumatism, and the treatment should not be abused in such cases.

Investigations with the objective to improve treatment for rheumatoid arthritis are increasing each year, and progress is being made. In the controversial questions that surround gold therapy, one should not be prejudiced, but should instead have an open mind and stand ready to be influenced by new information. As improvements are developed they should be put into practice. If gold salts with more dependable therapeutic value and less toxicity, or an improved schedule of administration of gold compounds are developed, these improvements should be substituted for the recommendations made in this chapter. Improvements in treatment for rheumatoid arthritis may develop so as to eliminate need for consideration

suspect that BAL might be beneficial in cases of gold intoxication. This has been demonstrated to be true both by animal experiments and by clinical trial.

The considerable investigation[63, 86] concerning the manner of action of BAL leads to the following reasonable explanation. It is postulated that toxic reactions occur when gold is in combination with tissue protein through a sulfhydryl linkage, that BAL competes successfully with the tissues for gold, and that the inert gold—BAL complex is more stable than gold—tissue complex so that the gold retained in the body is rendered non-toxic.[14] There is slight increase in urinary excretion of gold for a short while after treatment with BAL,[63] but this is probably unimportant in combating gold toxicity.

Several investigators[37 51 53] have shown that BAL prevents death of rats injected with otherwise lethal doses of gold salts, and reduces pathologic effects of sublethal doses. The earlier BAL is administered after the injection of gold salts the more effective it is in preventing or combating the gold intoxication.

Two, three-dimercapto-propanol has been shown to be effective clinically in the treatment of gold toxicity in various forms[15] including: exfoliative dermatitis,[10 57 68] thrombopenic purpura,[65] granulopenia,[85] severe stomatitis,[67] and conjunctivitis.[67] From recent experience in several instances it appeared to arrest and reverse toxicity that seemed certain would terminate fatally without the use of BAL. Clinically, as in animal investigations, the earlier BAL is used after toxicity develops the more beneficial it is; if toxicity has existed many months it has often failed to be beneficial, for reasons at this time unexplained. BAL is prepared in a 10 per cent solution of benzyl-benzoate in peanut oil, for use intramuscularly. For quite severe intoxication, it needs to be used vigorously in order to have a dependable effect. It has been shown[63] that 80 per cent of the BAL injected intramuscularly appears in the blood within an hour and that the maximum concentration in the blood exists between one and three hours after injection; it is almost completely excreted within twenty-four hours. In all types of [illegible] effective when injected at four hour [illegible] the first and second days [illegible] days thereafter. If marke[illegible] within a few days after start of BAL therapy, it may be given only once daily from the fifth to the eighth or tenth day of treatment. The dose used for each injection is the same and is calculated on the basis of body weight: 2 5 mgm. of BAL (0 025 cc solution) per kgm.

There may be undesirable side effects of BAL. These include malaise, nausea, vomiting, salivation, lacrimation, blepharospasm, paresthesia, [illegible] pain in the extremities or abdomen and [illegible]

reactions are not serious in [illegible] pass off within a day after cessation of treatment because of the rapid elimination of BAL. Pain and cramps in the muscles of the extremities and sometimes carpal-pedal spasms, occasionally observed following injections of BAL, were accompanied by Chvostek's sign and lowered serum calcium, phosphorus and magnesium.[41] The most troublesome complica-

of gold therapy; in this case investigators who have busied themselves

of current research.

BIBLIOGRAPHY

1. ADAMS, C H., and CECIL, R L · Ann Int Med, 33, 163, 1950.
2 BATTERMAN, R C., TRAEGER, C H, and WAINERDI, H.. Report at Meeting of New York Rheumat Assn, April, 1952
3. BLOCK, W. D. and BUCHANAN, O H. J Biol Chem, 136, 379, 1940
4 ——— Jour. Lab. and Clin Med, 28, 118, 1942.
5. BLOCK, W. D., BUCHANAN, O H. and FREYBERG, R H.· Jour. Pharmacol and Exper Med, 76, 355, 1942
6 BLOCK, W D, GEIB, N C. and ROBINSON, W. D · Proc Am. Fed. Clin Research, 6, 239, 1947.
7. BOLAND, E F. and HEADLEY, N E Report, Annual Meeting, Amer. Rheum Assoc, Chicago, Ill, 1948
8 BOOTS, R H, COSS, J H, JR, and RAGAN, C.· Trans Assoc. Am Physicians, 60, 259, 1947
9 BROWNING, J S, RICE, R M, LEE, W V and BAKER, L. M · New England Jour Med, 237, 428, 1947
10 BURNETT, J. A · Brit Med Jour, 2, 670, 1947.
11 BURNETT, J C. *Gold as a Remedy in Disease, London, The Homeopathic Publishing Company, 1879*
12 CECIL, R L, KAMMERER, W H and DEPRUME, F J.· Ann Int. Med, 16, 811, 1942
13 COHEN, A, GOLDMAN, J and DUBBS, A W · New England Jour Med, 233, 199, 1945
14 ———. Jour. Am Med Assn., 133, 749, 1947.
15 COTTELL, McK, *et al* New York State Med Jour, 48, 643, 1948
16 Council on Pharmacy and Chemistry· New and Non-official Remedies, Phila-
17.
18
19 ...er Therap, 69, 350, 1940
20 DAWSON, M H, BOOTS, R H and TYSON, T L: Tr Assoc Am. Physicians, 56, *330, 1941*
21. DIENES, L.· Jour Inf. Dis, 65, 24, 1939
22 DIENES, L, BERG, R L and WEINBERGER, H. J.. Report Annual Meeting Am
23 ... Jour, 2, 314, 1940
24
25 ... Brit. Jour. Exper Path, 21, 13, 1940
26 FRASER, T N: Ann. Rheum Dis, 4, 71, 1945
27 FORESTIER, J Bull et mem Soc méd d hôp de Paris, 53, 323, 1929
28 ———· *Jour. Lab and Clin Med, 20, 827, 1935*
29 FREYBERG, R. H Proc Staff Meet, Mayo Clin, 17, 534, 1942
30 ———· Unpublished observations
31. ... Jour. Clin Invest, 20, 401, 1941
32
33 ... W S.: Unpublished data pre... ll, 1943.
34 ... S · Clinics, 1, 537, 1942
35
36 ... hiladelphia, W. B Saunders Co, 136, 1913
37. GILLMOR, S and FREYBERG, R. H: Jour. Lab. and Clin Med, *33, 1024, 1948*
38. GOLD, H: Am. Jour Med, 4, 1, 1948

tions of treatment with BAL are the considerable pain which usually attends the injection and abcesses which occasionally develop at the injection site. Many abscesses have been sterile, but some contain stalphylococci and on one occasion a staphylococcus bacteremia lasted many days after the development of a staphylococcus abscess at a site of injection of BAL. Often, injections must be made through abnormal skin, where organisms appear to be more abundant and may be introduced into the muscle by the needle Because of the ease of abscess development at site of injections, meticulous sterilization of skin is advised

With the advent of cortisone and corticotropin other good antidotes for gold toxicity have become available Different forms of dermatitis due to gold have been quickly and completely eliminated by these hormones, and this is to be expected because of the allergic nature of this type of toxicity [illegible]

agents for such reactions to gold The treatment of choice among these preparations, currently, is corticotropin used by slow intravenous drip in doses of 25 mg, daily for several days Frequently treatment is required for only a short while, although recurrences may develop for which reinstitution of treatment is necessary If treatment is required for many weeks, which is infrequent, slow-absorbing hormone intramuscularly, is preferable Cortisone, orally or intramuscularly, is not as dependably effective as is corticotropin, but in many cases it has very satisfactorily relieved the dermatologic manifestations of gold toxicity and in cases requiring prolonged use of the antidote it has the attractiveness that it can be administered orally As is true with BAL, the early toxic reactions usually respond more favorably than those that have been long-existing Further details regarding corticotropin and cortisone will be found in Chapter 17 (Chapter on Cortisone and ACTH)

Because many toxic reactions from gold may be mild and short in duration it does not seem wise to begin the use of BAL, cortisone or corticotropin at the beginning of gold intoxication Close observation of the patient should be made, and if toxicity becomes rapidly worse and quite severe, one of these antidotes should be used promptly for best results are obtained when it is used early in the course of intoxication If toxic manifestations recur after they have been effectively suppressed the antidote should be used again

Effect [illegible] ACTH or BAL, properly [illegible] toxicity relieves the anxie[illegible] Even so, precautio[illegible] expected, for preventing or minimizing toxicity is far better than treating severe toxicity Generally, when there is a severe toxic reaction from gold, especially generalized dermatitis, the arthritis usually is greatly improved and frequently completely arrested This has been so consistently observed that some physicians believe the benefit from gold salt therapy is due to the toxic reaction and that it is desirable to produce clinical toxicity to accomplish best results. With this reasoning, the author cannot agree, for, excellent benefit from gold occurs commonly without any toxic manifestations Every effort should be made to treat effectively and avoid serious toxicity. It seems a

39. Goldie, W · Ann. Rheum. Dis, 1, 319, 1939
40. Graham, J W. and Fletcher, A. A · Canad Med. Assoc Jour, 49, 483, 1943
41. Green, B and Russel, B F · Proc Roy Soc Med, 41, 110, 1948
42. Hartfall, S J, Garland, H. G. and Goldie, W.: Lancet, 2, 784 and 838, 1937
43. Hartung, E F.· Bull New York Acad Med, 19, 693, 1943
44. Hartung, E. F and Cotter, J · J Lab and Clin Med, 26, 1274, 1941
45. Heilman, F R.· Science, 91, 366, 1940
46. Holbrook, W P. and Hill, D F · Jour Am Med Assn, 107, 34, 1936
47. Kammerer, W L., Cecil, R. L., and Robbins, C · Report at Meeting of New York Rheumat Assn, April, 1952.
48. Key, J A, Rosenfeld, H. and Tjoflat, O E · Jour. Bone and Joint Surg, 21, 339, 1939
49. Kling D H, Sashin, D and Spanbock, J · Jour Lab. and Clin. Med, 24, 1241, 1939
50. Koch, R · Deutsch med Wchnschr, 16, 756, 1890
51. Kuzell, W C, Pillsbury, P L and Gellert, S A · Stanford Med Bull, 5, 107, 1947
52. Lande, K · Munchen med. Wchschr, 74, 1132, 1927
53. Levey, S and Smyth, C J · Jour Lab and Clin. Med, 32, 1364, 1947
54. Libenson, L · Unpublished data
55. Lockie, L M, Norcross, B M and George, C W · Jour Am Med Assn, 133, 754, 1947
56. Logefeil, R C and Hoffman, R A · Journal-Lancet, 61, 221, 1941
57. Margolis, H M and Caplan, P S · Ann Int Med, 27, 353, 1947
58. Margolis, H M, and Caplan, P S · Jour. Am Med Assn, 145, 382, 1951
59. Mollgaard, H · Chemotherapy of Tuberculosis, Nyt Nordisk forlag, Kjobenhaon, Arnold Busck, 1924
60. Oren, H · Jour Med Soc New Jersey, 33, 591, 1936
61. ——— Med Rec, 159, 420, 1946
62. Patterson, M, and Traeger, C H · Report at Meeting of New York Rheumat Assn, 1951
63. Peters, R A *et al* · Biochem Jour, 41, 370, 1947
64. Phillips, R T · New England Jour Med, 214, 114, 1936
65. Pick, E · Wien klin Wchnschr, 40, 1175, 1927
66. Preston, W S, Block, W D and Freyberg, R H · Proc Soc Exper Biol and Med, 1, 253, 1942
67. Price, A E and Leichtentritt, B · Ann Int Med, 19, 70, 1943
68. Ragan, C and Boots, R H · Jour Am Med Assn, 133, 752, 1947
69. Ragan, C and Tyson, T L · Am Jour Med, 1, 252, 1946
70. Rawls, W B · New York Med, 3, 19, 1947
71. Rawls, W B, Gruskin, B J, Ressa, A A., Dworzan, H J and Schreiber, D · Am Jour Med Sci, 207, 528, 1944
72. Robinson, D · Canad Med Assoc Jour, 1, 223, 1944
73. Rose, P A · Illinois Med Jour, 92, 175, 1947
74. Rothbard, S, Angevine, D M and Cecil, R. L · Jour Pharmacol and Exper Therap, 72, 164, 1941
75. Sabin, A B · Bact. Rev, 5, 1, 1941
76. Sabin, A B and Warren, J · Jour Bact., 40, 823, 1940
77. Sashin, D, Spanbock, J and Kling, D H · Jour Bone and Joint Surg, 21, 723, 1939
78. Short, C L., Beckman, W W and Bauer, W · New England Jour Med, 235, 362, 1946
79. Smyth, C J and Freyberg, R H · Univ (Michigan) Hosp Bull, 7, 45, 1941
80. Snyder, R G, Traeger, C H and Kelly, L · Ann Int Med, 12, 1672, 1939
81. ——— New York State Jour Med, 43, 245, 1943
82. Spray, G H, Stocken, L A and Thompson, R. H S · Biochem Jour, 41 362 1947
83. Stengel, F · Bull Hosp Joint Dis, 5, 114, 1944
84. Sundelin, F · Nord med, 37, 303, 1948
85. [illegible] Ann Int Med, 36, [illegible], 1952
86. [illegible] Biochem Jour, 41, 342, 1947
87. [illegible]
88. [illegible] Calif Med Jour, 66, [illegible], 1947

justified compensation, however, that the patient who has the unpleasantness accompanying gold toxicity should have the reward of relief from the arthritis during this time, and that is the rule.

Some European physicians[84] have reported so much lower toxicity using gold compounds with the same chemical characteristics that are used in the United States, that it has led to the question whether or not there is a difference in preparation which may be an important factor in toxicity. No knowledge to substantiate this suspicion has appeared; it seems unlikely.

Reinstitution of Gold Therapy after Toxicity.—The continuance or *recurrence of active joint disease after recovery from gold toxicity* sometimes prompts the cons[illegible] who has had toxicity. [illegible] this problem. It can b[illegible] has had a severe form of intoxication from gold *should never* have treatment with gold compounds thereafter, for experience has shown that toxicity will most certainly develop again. Oftentimes, only one or two small doses of gold salt in a patient who has had toxicity from gold may cause unusually severe and prolonged toxic manifestations. Therefore, after the occurrence of severe dermatitis, enteritis, hepatitis, nephritis or blood dyscrasia due to toxic effects of gold, subsequent use of gold compounds should not be attempted. In some patients with mild dermatitis or stomatitis it has been possible to reinstitute gold therapy soon after abatement of the toxicity. [illegible]

[illegible] doses of gold salts indefinitely when the customary (larger) doses cause toxicity. Hence, in patients who have severe, progressive arthritis for whom the use of gold salts is again strongly desired, careful trial is justified, providing the necessary precautions are taken, and if previous toxicity has been mild.

RELAPSE FOLLOWING GOLD THERAPY

When the course of many patients is followed for several years, frequently [illegible] with gold salts, [illegible] ment gives way to [illegible] curs a few months [illegible] instances relapse [illegible] of permanency of [illegible] res and represents [illegible] ion Incidence of relapse has been reported variously from 7 to 75 per cent with an average of 25 per cent (Table 8). In those who had remissions, and in those who had improved greatly but whose disease had not become completely arrested during gold therapy, 50 per cent relapsed after treatment was stopped Ragan and Tyson[69] report the highest relapse rate; of 142 cases

CHAPTER 17

CORTICOTROPIN, CORTISONE, AND HYDROCORTISONE

By RICHARD H. FREYBERG, M.D.

ONCE in a physician's lifetime a "scientific bombshell" may explode—the discovery of a fact or the novel application of a principle, old or new, which may revolutionize the conception of medical problems, point a new approach to the solution of perplexing puzzles of physiology or pathology, or open the way to superior treatment of illnesses in a large area of the medical field. Such an explosion occurred in the spring of 1949 in Rochester, Minnesota, with the announcement by Hench, Kendall, Slocumb and Polley[15] of the remarkable effects of cortisone (then known as Kendall's Compound E) and corticotropin (pituitary adrenocortico-tropic hormone, "ACTH"). This event was, in the medical world, comparable to the discovery and application of nuclear fission in the world of physics, an event which literally pushed many widely diversified medical activities into a new era in this "atomic age." All the more remarkable, and at the same time welcome, it was that this cardinal advance was launched in the area of rheumatic diseases—a sector of the medical field which for so long has been baffling to both the investigator and the clinician. Knowledge based upon this recent advance has spilled over rapidly into many other parts of the large field of medicine, indeed, on some of the non-rheumatic problems the new light may shine as brilliantly and may reveal as many hitherto hidden corners as it is expected to do in the network of highways and bypaths in the province of rheumatism. But our present interests are confined to the latter scope.

[illegible]

tion and supply of these materials depend. Truly, the medical profession acknowledges with compliments and gratitude, the remarkable work of the chemists, physicists and pharmaceutical engineers whose phenomenal perfection of methods for synthesis and production of the materials allowed the medical investigator and the clinician to study their effects.

The volume of research that has resulted from this beginning in 1949 is huge. Investigations have been pursued at a feverish pace so that the complexion of many medical subjects affected by these studies keeps changing. Still, in this ever-changing field, it is necessary to take stock of the situation at certain times, and this is one such occasion. That important developments may alter, significantly, the evaluation from the time of preparation to the time of appearance of this inventory, is a risk keenly appreciated by the writer, and it is his hope that the reader also will be aware of this possibility. In such a spirit this difficult but interesting task is undertaken.

followed for three years, 75 per cent relapsed The relapse instead of progress occurred as early as six weeks and as long as two years after cessation of treatment In a small group of 21 patients, Short[74] found 62 per

graphic representation of the sedimentation rate in the same patient whose earlier course is shown in Figure 86. Notice that several months after complete arrestment of the arthritis a relapse occurred with sharp elevation of the sedimentation rate. The patient was unable to continue his work and normal living which he had enjoyed for the first time since the onset of arthritis several years before Gold therapy was reinstituted and accompanied by another remission wherein the improvement was slower than

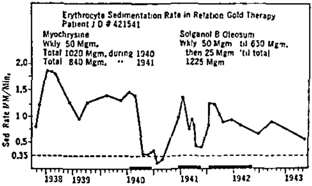


FIG 89 —The continued graph showing the erythrocyte sedimentation rate of the same patient in whom the sedimentation rate during the first months of observation is shown graphically in Figure 86

was observed with the first course of gold therapy, but it did not progress to complete arrestment of the disease Again the patient returned to work, and treatment with gold salt was discontinued, again there was a relapse followed by remission after further treatment, but with less improvement than occurred during either previous treatment period

Such experiences have been observed in many patients Fortunately, the relapse has been found by most observers to be less severe in the majority of patients than the original disease Fortunate also is the fact that in approximately 80 per cent of patients who relapse gold therapy again produces remission It is the rule that the speed and degree of improvement is not as great with subsequent treatment as had occurred with initial treatment.

The frequency of relapse has led to different treatment schedules aimed to reduce this disappointing experience (to be discussed below).

METHOD OF TREATMENT

Having become thoroughly familiar with the nature of gold salt therapy, especially in regard to the metabolism of gold, its potential therapeutic

THE NATURE OF CORTISONE AND CORTICOTROPIN

Cortisone (17-hydroxy,11-dehydrocorticosterone) is one of many steroids* originally obtained by extraction from adrenal glands. As these steroids were isolated and identified they were designated by Kendall as Compounds A, B, . . etc. Later, several of these were partially synthesized from several different precursors obtained from animal or plant sources The first method for partial synthesis employed desoxycholic acid [illegible]

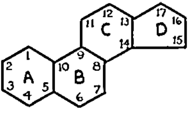


FIG 90 —The chemical nucleus of the adrenal cortical steroids.

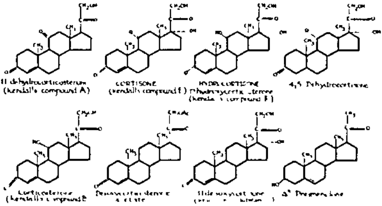


FIG 91 —Structural formulæ for cortisone and several related steroids

The nucleus of the adrenocortical steroids is shown in Figure 90 This structure can be varied by changes in the ring system (A, B, C and D) and in the side chains The formulæ for several of these steroids, more important in scientific investigations are shown in Figure 91 Note the striking similarity of these steroids' One may differ from another only by a single atom—for instance, the structure of cortisone is exactly the same as that of hydrocortisone (Compound F) except that the latter steroid has

* The term steroid' is a generic name for certain tetracyclic compounds Some steroids occur naturally, some do not Widely distributed in nature are sterols tetracyclic compounds which are monohydroxy alcohols of steroids of this group cholesterol is the best known

value and toxicity, the limitations of its usefulness, and the precautions re-

treatment is very important, and can be more wisely made because of re-

treatment of rheumatoid arthritis.

Gold Sodium Thiosulfate.—This inorganic salt of gold and sodium is water soluble, contains 37 per cent gold, is prepared both as a powder to be dissolved prior to injection and as a solution ready for injection, in ampules containing appropriate single doses It can be injected either intramuscularly or intravenously. Commercial preparations include *Sanocrysin*, *Sanochrysine*, and *Crisalbin*.

Gold Sodium Thiomalate.—For years this aqueous soluble salt has been prepared in ampules of convenient dosage, ready for injection. This salt is to be injected only intramuscularly. It is 50 per cent gold. The trade name is *Myochrysine*

Gold Thioglucose.—This water soluble salt of gold has been used to a small extent in solution, known as *Solganal*. More commonly it has been employed as a suspension in oil for intramuscular injection. It is a salt which contains 50 per cent gold Ampules containing commonly used single doses and multiple dose vials are available.

Gold Thioglucoamide.—This is an insoluble gold salt in oil suspension for intramuscular injection. Vials containing enough for many injections are available *The salt is 54 per cent gold. It is marketed under the name of Lauron*

Other compounds less commonly employed in the United States include:

Gold sodium allyl-thio-urea benzoate, a salt in aqueous solution which can ... The trade name is *Lopion*.

... salt with 50 per cent gold ... amuscular injection. It is known as *Parmanil*.

... *riphal*. ... n as *Auro*- ... only 5 per ... y a solution of which 1 cc. contains 25 mgm of gold known as *Aurocein-25* has been prepared

Gold sulfide, a simple, insoluble, inorganic salt containing 86 per cent gold prepared in colloidal suspensions of 1 and 2 per cent (salt content), for intravenous, intramuscular and oral administration It has been sold as *Aurosulfide*.

Colloidal gold preparations of various (small) gold content, for use intravenously, intramuscularly and orally.

CHAPTER 17

CORTICOTROPIN, CORTISONE, AND HYDROCORTISONE

By Richard H Freyberg, M.D.

Once in a physician's lifetime a "scientific bombshell" may explode—the discovery of a fact or the novel application of a principle, old or new, which may revolutionize the conception of medical problems, point a new approach to the solution of perplexing puzzles of physiology or pathology, or open the way to superior treatment of illnesses in a large area of the medical field Such an explosion occurred in the spring of 1949 in Rochester, Minnesota, with the announcement by Hench, Kendall, Slocumb and Polley[18] of the remarkable effects of cortisone (then known as Kendall's Compound E) and corticotropin (pituitary adrenocortico-tropic hormone, "ACTH") This event was, in the medical world, comparable to the discovery and application of nuclear fission in the world of physics, an event which literally pushed many widely diversified medical activities into a new era in this "atomic age." All the more remarkable, and at the same time welcome, it was that this cardinal advance was launched in the area of rheumatic diseases—a sector of the medical field which for so long has been baffling to both the investigator and the clinician. Knowledge based upon this recent advance has spilled over rapidly into many other parts of the large field of medicine, indeed, on some of the non-rheumatic problems the new light may shine as brilliantly and may reveal as many hitherto hidden corners as it is expected to do in the network of highways and by-paths in the province of rheumatism But our present interests are confined to the latter scope

[illegible]

of the equally [illegible]
tion and supp[illegible]
acknowledges [illegible]
chemists, physicists and pharmaceutical engineers whose phenomenal perfection of methods for synthesis and production of the materials allowed the medical investigator and the clinician to study their effects

The volume of research that has resulted from this beginning in 1949 is huge. Investigations have been pursued at a feverish pace so that the complexion of many medical subjects affected by these studies keeps changing Still, in this ever-changing field, it is necessary to take stock of the situation at certain times, and this is one such occasion. That important developments may alter, significantly, the evaluation from the time of preparation to the time of appearance of this inventory, is a risk keenly appreciated by the writer, and it is his hope that the reader also will be aware of this possibility. In such a spirit this difficult but interesting task is undertaken.

THE NATURE OF CORTISONE AND CORTICOTROPIN

Cortisone (17-hydroxy,11-dehydrocorticosterone) is one of many steroids* originally obtained by extraction from adrenal glands As these steroids were isolated and identified they were designated by Kendall as Compounds A, B, . etc. Later, several of these were partially synthesized from several different precursors obtained from animal or plant sources The first method for partial synthesis employed desoxycholic acid obtained from ox bile as the starting substance Later, methods for partial synthesis of these steroids were developed from other sources including the Mexican yam, saponins and certain digitalis derivatives

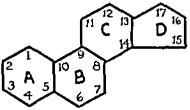


FIG 90 —The chemical nucleus of the adrenal cortical steroids.

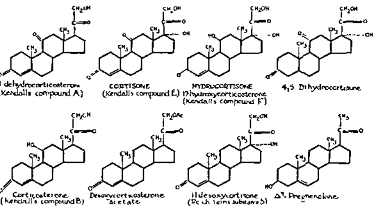


FIG 91 —Structural formulæ for cortisone and several related steroids

a single atom—for instance, the structure of cortisone is exactly the same as that of hydrocortisone (Compound F) except that the latter steroid has

* The term "steroid" is a generic name for certain tetracyclic compounds Some steroids occur naturally, some do not Widely distributed in nature are sterols, tetracyclic compounds which are monohydroxy alcohols of steroids, of this group, cholesterol is the best known

effect. None of the known metabolic or hormonal actions of cortisone can explain the manner of production of the anti-rheumatic effect. The speed of response (remarkable diminution of stiffness and pain, allowing great increase in movement of joints that are still inflamed—within a few hours) strongly suggests that the benefit is obtained at the connective tissue level through a beneficially altered enzyme system But what this is, and how it is altered—if it, indeed, is the mechanism—is not known. Other possibilities include a change in antigen-antibody relationship or alteration in tissue permeability. *The basic cause of the inflammation is not eliminated by cortisone* as shown by the tendency for the activity of the disease to return after cortisone administration is discontinued. Thus, the result of cortisone administration is seen to be *suppression but not an elimination of the pathologic state*

Dangers.—In using cortisone or corticotropin to accomplish a clinical anti-rheumatic effect, any of the other physiologic effects of these hormones *may produce complicating problems ("side-reactions," "toxic-effects")* These may be trivial, or may be of such serious magnitude as to preclude continued use of the hormone. The nature of these complications can be readily appreciated from a review of the physiologic effects of the hormones. The undesirable features of *Cushing's syndrome* may be troublesome—obesity, moon-face, hirsutism, acne. *Edema* may present a problem, especially if it involves the lungs as it may when increased blood volume may contribute to congestive heart failure The excretion of potassium may result in a state of *potassium deficiency* in the cells, with serious consequences. Severe *osteoporosis* has been the basis for fractures which occur after only slight trauma. Carbohydrate metabolism may be so disturbed that problems of "*diabetes*" are encountered. Suppression of ovarian function may lead to very annoying troubles of *menopause*. Many effects on the *blood* and *circulation* have been encountered

somnia, and psychoses characterized by excitement and agitation, or by depression.

Experience has shown that complications are more frequent and may be more severe in females, especially those in or near menopause. This has become a matter of practical importance

Inactive tuberculosis may become activated due to the inhibiting effect of cortisone on fibrosis.

an hydroxyl (OH) radical attached at carbon eleven instead of oxygen which is present in cortisone With only such small differences in composition these steroids may possess greatly different chemical, physical, and (extremely important in medic[illegible]

As a group, these steroids c[illegible] absence of an O atom at the C[illegible] known as the *oxy-corticoster*[illegible] corticosteroids. There are certain general pharmacologic properties attached to these classes as will be noted later.

Cortisone is secreted by the adrenal cortex in small amounts in relation to the larger quantity of hydrocortisone, the most abundantly secreted cortical steroid. When prepared by partial synthesis, these steroids are white or creamy white, odorless crystals or crystalline powder. They are relatively insoluble in water and physiological solutions, so that for injection purposes, microcrystals are suspended in an aqueous medium. Such suspensions are quite stable at room temperature.

Corticotropin is the adrenocorticotropic hormone (ACTH) secreted by the anterior lobe of the pituitary gland. It is obtained by extraction from the pituitary glands of domestic animals The majority of this hormone *available for use is obtained from the hog pituitaries for the yield is largest* [illegible]

rived from the extracted hormone. These peptides have been shown to possess the same biologic activity as their progenitor It is hoped that further chemical studies may clarify the chemical nature of corticotropin, and that its synthesis may be accomplished. Currently the entire supply of corticotropin depends upon the available supply of animal pituitaries

Corticotropin is prepared in the form of lyaphilized crystals to be dissolved in water. The concentration of the solution can be varied for convenience of injection. In dry form, the preparation is quite stable especially at refrigerator temperature; recently stable solutions have been prepared

THE PHYSIOLOGIC AND PHARMACOLOGIC ACTIONS OF CORTISONE AND CORTICOTROPIN

The end biologic effects of cortisone and corticotropin are essentially the same, but their route of action is decidedly different (Figure 92). The action of corticotropin is directly on the adrenal cortex which it stimulates to discharge a larger quantity of preformed cortisone, hydrocortisone and other steroids. Corticotropin is ineffectual unless it reaches the adrenal glands in an adequate amount, and unless the adrenals are capable of responding to the stim[illegible] conditions do not obtain identically in all individuals, [illegible] is probably directly u[illegible] pendent of the adrenal glands. Cortisone added to the body by [illegible] or ingestion apparently needs only to reach the tissues to produce its physiologic actions which are limited only by the effective dosage of the steroid. Cortisone administered to a patient produces the effect of this steroid, whereas the result of corticotropin is the sum of effects of all of

A complication peculiar to corticotropin and not encountered with the use of cortisone is the development of hypersensitivity to this protein substance. Although this rarely occurs, severe anaphylactic shock, or other frightening and serious sensitivity reactions, may need to be reckoned with.

All of the undesired effects of cortisone or corticotropin are temporary in nature and produce problems only during the continued use of the hormone. All are reversible after discontinuing use of the hormones. This is true, also, with the adrenal, pituitary and thyroid depression that may occur during prolonged use of cortisone. Recent studies of experimental animals have shown that atrophy of the adrenal glands is not permanent, and ... *The fear of*

... hort duration, cortisone and corticotropin can be expected to benefit the patient greatly and because of the short period the hormone is required, little or no difficulty ...

... The details and ... cotropin for each rheumatic disease will be in some instances discussed elsewhere in connection with the management of specific illnesses, but it will be advantageous to note here certain variations in the clinical response of different rheumatic diseases.

RESPONSE IN ACUTE RHEUMATIC DISEASES

Rheumatic Fever.—Many investigators[2,3,25,29,44] have reported excellent anti-rheumatic effects of cortisone and corticotropin in patients with acute rheumatic fever. Within twenty-four or forty-eight hours signs of illness lessen or completely disappear, there is a marked sense of increased well-being—the feeling of "toxicity" improves, and weight increases. Signs of ... movement of the joints ... plete ... dings (erythrocyte sedimentation rate, electrocardiogram) usually become normal more slowly but are completed within four to eight weeks. If treatment is discontinued gradually, many patients remain well, should the disease again become active, a second or third course of treatment will usually effect suppression which will be sustained. In this acute illness treatment should be initiated in adequate amount as soon as the disease can be identified, in order to suppress the inflammatory process until the disease activity would subside under natural influences, and in this way hope to minimize the cardiac damage. Whether cardiac protection results is not ...

who states "... it is hoped that the ... the disease can be forestalled."

How the effects of cortisone and corticotropin treatment for rheumatic fever compare with those of salicylate therapy has been a matter of great

the steroids secreted by the adrenal cortex, hence may differ somewhat

These are summarized in Table 14.

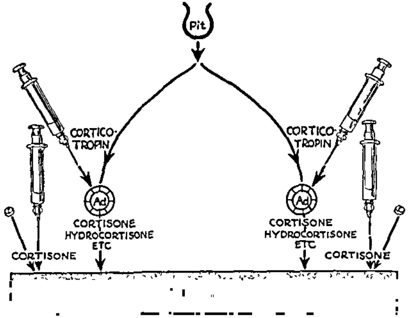


FIG 92 —Schematic drawing to show different routes of action of corticotropin and cortisone Corticotropin acts to stimulate the adrenal cortex to discharge more cortisone, hydrocortisone, and other steroids which then produce their effects in the tissues Similar effects result from the injection of cortisone or hydrocortisone directly into the tissues, or by the ingestion of these steroids which are then absorbed and reach the tissues via the blood

Metabolic Effects —Increased amount of cortisone in the body whether administered directly or as a result of stimulation of the adrenal glands by injection of corticotropin causes numerous alterations in metabolic proc-

decrease the tissue potassium and be reflected by a decrease in serum potassium Calcium and phosphorus are excreted in larger than normal amounts, which if substantial in degree and continued over a long period of time may result in osteoporosis Among the effects on organic metabo-

interest, but this, too, will require many more years of careful study to clarify.

Treatment must be individualized in each case, but in general it is wise, if cortisone or corticotropin are employed, that the dose be sufficient to effect a quick and complete arrest of the disease, and for this effect, rather large doses of the hormone are usually required. Seldom is there more than nuisance difficulty from physiologic effects of the hormone other than the anti-rheumatic effect, for the period of time the hormone is required is relatively short. However, care must be taken to avoid potassium deficiency, edema and cardiac decompensation. (*See also* Chapter 32.)

Acute Bursitis.—This rather minor and usually self-limited illness may at times be difficult to control by conservative measures and, because of

movement is regained Seldom is treatment required for longer than two or three weeks, during which time complications seldom occur. (*See* Chapter 54)

If the bursal inflammation continues and becomes a chronic problem, or if tendonitis and/or periarthritis follow the acute bursitis (as not infrequently is the case especially at the shoulder) hormone therapy occasionally may be employed in order to relieve the pain and stiffness so that physical treatment can be used to increase the range of motion Two to three weeks of rather intensive hormone administration usually suffices to accomplish maximum help in this situation *The dangers of hormone administration should be considered carefully, however, before using in any mild or localized condition*

Acute Gouty Arthritis.—Corticotropin quite consistently suppresses a

of gout for those patients who are intolerant of, or unresponsive to these simpler chemotherapeutic agents. (*See* Chapter 41)

USE IN RHEUMATOID ARTHRITIS

Since the desire for, and the problems of use of cortisone or corticotropin are greatest in rheumatoid arthritis, these considerations deserve careful attention The practical questions are (1) Can cortisone and corticotropin be used safely and effectively to benefit patients with rheumatoid arthritis? and (2) if so, what therapeutic schedule is best[9] Experiences of the writer and his collaborators over three and one-half years help to answer these questions.[12, 13]

Three possibilities exist for the beneficial use of these hormones for rheumatoid arthritis:

TABLE 14

EFFECTS OF CORTISONE

Metabolic	*Endocrine*	*Cytologic*	*Immunologic and Serologic*	*Enzymologic*	*Neuromuscular*
Inorganic Sodium retention Chloride retention Water retention Potassium excretion Calcium excretion Phosphorus excretion *Organic* Increases glyconeogenesis. Increases glycogen deposits in liver Opposes action of insulin in periphery. May elevate blood sugar Lowers renal threshold for glucose. Increases absorption and storage of fat. Increases catabolism of protein. Increases excretion of nitrogen, uric acid and creatinine	Increases insulin requirement May decrease, delay or completely eliminate menstruation. May depress thyroid function Decreases adrenal cortical function and corticotropin secretion in pituitary	Decreases Eosinophils. Lymphocytes. Increases Production or release of reticulocytes. Erythrocytes Neutrophils Inhibits fibroplasia May (in large doses) retard wound healing May reactivate ulcers. Causes involution of lymphoid tissue in some lymphoblastomas.	Decreases serum globulin. Increases serum albumen. Diminishes antibody response (under certain conditions). Decreases tuberculin reaction Reduces or prevents tissue irritation from some chemicals Diminishes (in large doses) localization of infections. Suppresses clinical manifestations of infection.	Decreases hyaluronidase activity. Increases Pepsin Trypsin Decreases Lysozyme.	Increases muscle strength and work May increase psychomotor activity May produce exaggerated sense of well-being May precipitate psychoses Excitation Depression May alter electroencephalogram, increase frequency of alpha waves, or appearance of slow rhythms

1. Repeated administration of cortisone or corticotropin for relatively short periods [illegible] without hormones.

[illegible] these hormones and thereafter sustain

3. To use cortisone or corticotropin for prolonged periods as "maintenance treatment"

When the first plan was employed—"course treatment" or "interval therapy"—it was hoped that undesirable side-effects would be eliminated and that adrenal and pituitary glands *might not* undergo functional suppression, and that with successive courses of treatment step-wise improvement in the arthritis *might be obtained.* However, in seventeen patient-experiences with cortisone and in fourteen patient-experiences with corticotropin, although significant benefit always occurred during use of the hormone, in *all* patients relapse promptly ensued after the hormone *was stopped*, and in no case was the relapse less in degree after successive courses of treatment (Fig. 93).

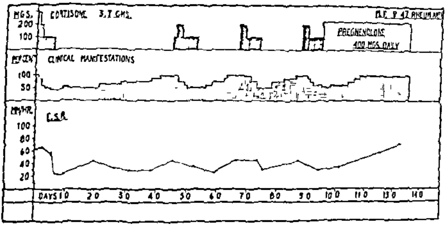


FIG 93 —A graphic representation of the inadequacy of repeated short periods of cortisone treatment in a patient with rheumatoid arthritis, and showing the failure of pregnenolone to sustain cortisone improvement. (Freyberg *et al.*, courtesy of Annals Rheumatic Diseases, Brit Med Assn)

Likewise, when the second plan was used, similarly disappointing results were obtained Many substances were used in an effort to sustain the benefit initially obtained by cortisone or corticotropin. These included some common steroids used for other purposes, and many newly synthesized steroids, all having chemical structures very similar to that of cortisone. Many trials were made with pregnenolone. It was hoped that these steroids might have some anti-rheumatic effect which, even though weak, might be sufficient to sustain improvement effected by the cortisone or corticotropin, and that they would have no undesirable effects. Although well tolerated in most instances, none gave dependable benefit. Others[33] have had essentially the same experience (*See also* Chapter 15.)

It seemed reasonable to expect that gold salts administered after these hormones, or overlapping the latter weeks of their use, and continuing

lism are. an increased catabolism of protein resulting in a negative nitrogen balance and wasting of muscle tissue. The serum albumen is increased when it had been below normal, and the serum globulin is decreased if it were elevated. Uric acid excretion is increased, as is creatinine. The effectiveness of insulin in the periphery is inhibited, carbohydrate combus-

Endocrine Effects.—Cortisone is stored or metabolized for the most part, but some (estimated to be about 10 per cent) is excreted in the urine as corticoids, neutral reducing steroids, formaldehydogenic steroids or as 17-ketosteroids The cortisone which is retained in the body produces various inter-hormonal changes Menstruation usually is suppressed and may be completely inhibited with menopausal symptoms resulting Changes in the configuration of the body characteristic of Cushing's syndrome appear after prolonged cortisone administration—rounding (mooning) of

ommon:

in the

"need"

stimulus does not exist, and as a result the adrenal cortical function becomes suppressed and the output of pituitary corticotropin decreases. The function of the thyroid may be depressed

Cytologic Effects.—Characteristically there is a decrease in the number of circulating eosinophilic leukocytes, although this effect is quite variable and occurs at different speeds There is stimulation of bone marrow to increase the number of erythrocytes and neutrophils Lymphocytes are decreased and lymph tissue may be diminished

The blood is sometimes altered by the action of cortisone other than alteration of cells The blood pressure frequently is elevated, sometimes to hypertensive levels Prothrombin time may be decreased, enhancing thrombosis

Enzymatic Changes.—Cortisone has the ability to increase the production of pepsin and trypsin and to decrease the production of intestinal lysozyme Hyaluronidase activity is decreased by cortisone

Effect on the Central Nervous System.—The changes in the function of the nervous system are indefinite and unpredictable Euphoria is very common Nervous tension and an uncomfortable sense of "being driven" frequently occurs with larger doses of cortisone or corticotropin, as does insomnia Changes of mood occur and the psychomotor activity may become altered. Excitation or depression may occur and infrequently assume

tory" actions

Anti-allergic Action.—Many studies have suggested different ways in which

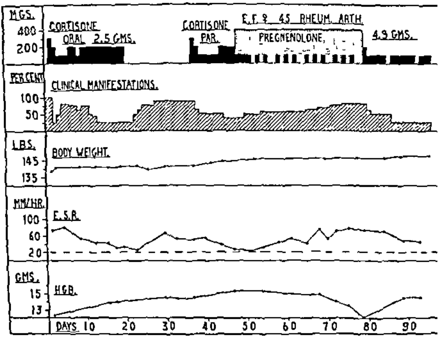
an alle

demon

end or

occurrence of nephritis in animals consistently appearing fourteen days

Traeger[46] has even reported results of studies which tend to show an antagonism between gold salts and cortisone or corticotropin when used simultaneously or in close sequence. There is also the disadvantage that the hormones might mask signs of intolerance for gold, a situation that might lead to severe toxicity after cortisone or corticotropin have been discontinued.



The many clinical trials by various investigators indicate that plan #2 provides generally unsatisfactory means of managing rheumatoid arthritis.

Although the prolonged use of cortisone and corticotropin (plan #3) presents many problems, it has been extensively studied.[4,11,12,13,19,47] It is readily appreciated that use of corticotropin, which requires injections either intravenously or intramuscularly is impractical treatment over many months' time, unless the patient is hospitalized. Hence, most studies of prolonged treatment have employed cortisone orally, after tablets became available. Earlier, when the steroid needed to be given intramuscularly, injections were required usually much less often than injections of corticotropin.

following intravenous administration of bovine gamma globulin, by injections of corticotropin, five days after injection of the antigen. However, antibody titers against the antigen, and reduction in compliment titers, were identical in the treated and untreated groups. It would appear from this, that the hormone produced its protective effect directly on the cell, rather than by interfering with antibody formation or antibody-antigen interaction. Rich[36] has shown essentially the same type of action from cortisone.

Anti-inflammatory Effects.—*Cortisone suppresses the inflammatory response* in tissues to practically all forms of irritation. It decreases the magnitude of the whole complex of inflammatory phenomena: vaso-

destruction or decreased local production of histamine. These reactions prevent the formation first of the usual lymphocytic barrier; then of the fibroblastic barrier to the injurious agent; thus, the establishment of natural lines of defense is suppressed. The net result of cortisone on injured or inflamed tissue is the tendency to minimize the reaction to the injurious agent while doing little to prevent its invasion of surrounding tissue.

In very high and unphysiologic dosage, the inhibition of proliferation of mesenchymal tissue including fibroblasts and their derivatives may interfere with connective tissue repair and scar formation as shown in animal experiments by Ragan and collaborators.[34,35] In doses commonly used clinically, this fibroblastic inhibition seldom is seen as an important action of cortisone.

The only other cortical steroid which has physiologic effects comparable to those of cortisone is hydrocortisone (Kendall's Compound F). This ... largely responsible for the effects

The physiologic changes produced by the injection of corticotropin are essentially those described for cortisone and hydrocortisone, with certain minor differences The reduction of circulating eosinophils is usually greater and occurs more consistently and at an earlier time than noted following injection of cortisone. Thus, the measurement of the degree of *eosinopenia* following a fixed technique has become a good index of adrenal-cortical stimulation and has become a commonly used laboratory guide for corticotropin effect.[42] Corticotropin stimulates the adrenal cortex to the elaboration of *all* of its steroid secretions so the result is that which comes from a mixture of many steroids and is not a pure "cortisone-effect." Sometimes, the use of corticotropin causes more salt retention or more nitrogen excretion than does cortisone given in an amount producing other effects that are equivalent. A consistent difference resulting from the administration of corticotropin as contrasted to cortisone is in the endocrine ... of the stimulation of corticotropin the adrenal cortices ... ecretion of corticotropin by ... sults from administration of

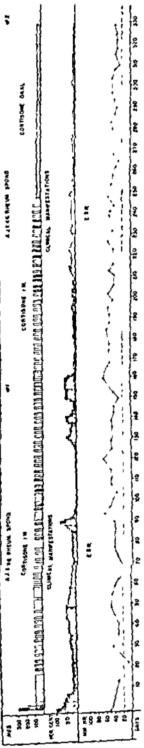


FIG. 95 —Showing the administration of cortisone orally as maintenance therapy, and an indication of the significant benefit that is often obtained thereby.

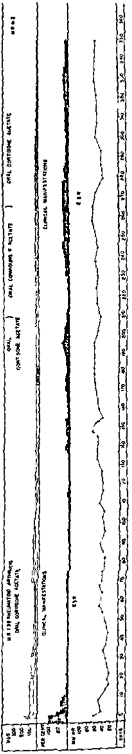


FIG. 96 —A graphic representation of the use of cortisone at first intramuscularly, then orally for prolonged treatment of a patient with rheumatoid spondylitis.

corticotropin are in most respects very similar to those produced by cortisone.

PREPARATIONS AND MANNER OF ADMINISTRATION

Cortisone.—Originally cortisone was prepared as a suspension of micro-

of two concentrations: 1 ml. containing 25 mg., and 1 ml containing 50 mg The next development was the demonstration that cortisone and cortisone acetate were effective when administered orally[10] Since then cortisone has usually been administered orally because of the greater convenience and lack of dependence of the patient upon others for injections

It was not surprising to find that this steroid is effective when given orally, for some other steroids are known to be, but it was surprising to find that the effect of equivalent doses was more rapid and shorter in duration after the oral administration compared to the intramuscular route The amount of oral cortisone required to accomplish a clinical effect comparable to that resulting from a certain intramuscular dose varies somewhat among different patients, but the average amount required orally is only slightly (about 20 per cent) greater than the amount needed parenterally to produce a comparable effect[11] The extensive studies of Thorn[22] comparing the physiologic and clinical effects of cortisone administered orally and intramuscularly indicate closely comparable results.

of administration

Best results are obtained when cortisone is given orally in divided doses, three or four times daily The effect is smoother and the drug efficiency is greater when it is given in divided doses Cortisone acetate is now prepared by several pharmaceutical manufacturers, and is supplied in tablets of 5, 10 and 25 mg sizes These tablets are scored for easy division into halves, thus allowing a wide variation of dose and administration

The technical and economic advantages of oral cortisone over intramuscular cortisone are obvious There is little variation among different patients in the effect of oral cortisone The presence or absence of HCl in gastric juice, and liver disease with significant functional impairment, have not altered the absorption or biological effectiveness of cortisone administered orally Abscesses which may occur at the site of intramuscular injection of cortisone acetate, of course, are obviated by using the steroid orally Nausea, vomiting and diarrhea may prevent dependable effects from oral cortisone The chief problem relating to the use of tablets of

hormone may be abused

Special preparations of cortisone acetate have been available for intravenous use So used, the steroid is rapidly effective and usually well

The common plan of prolonged or "maintenance" cortisone therapy has been about as follows: Ample hormone is administered to suppress the disease to a level which the patient and the doctor agree upon—the "desired effect"—after which a smaller dose of hormone is given daily to maintain this degree of benefit—the "maintenance dose." It is to be emphasized that the "desired effect" is *not complete suppression* of the disease which would require much too large doses of cortisone for main-

was administered.

treatment (more than 100 consecutive days) with cortisone was reviewed in 44 patients studied by the writer and his investigative group.[12] With such management, good to excellent anti-rheumatic effects were usually maintained *during* administration of the steroid In only 4 per cent of trials was long-term use of cortisone stopped because of poor therapeutic effect. In most instances a daily oral dose of 50 to 75 mg of cortisone acetate was required for "maintenance" If more than 75 mg. were required daily, it was found that complications sooner or later made it impossible or undesirable to continue treatment The variation in maintenance dose correlated chiefly with the severity of the disease, not with its duration or with the age, size or sex of the patient. In amounts up to 75 mg daily "maintenance" cortisone acetate was usually well tolerated, although the longer the treatment, the greater the chance for trouble

was stopped deliberately in order to study post-cortisone events In 6 instances (14 per cent) it was considered necessary to stop cortisone due to "diabetes" and mental depression (one instance of each), edema in 2 patients, and massive hemorrhage into the gastro-intestinal tract in 2 patients Five of these six patients were females

Just as the benefits *during* treatment were usually gratifying, the difficulties *after* prolonged treatment were very disappointing both to the patient and physician These events are summarized in Table 17. Within sixty days, relapse to pre-treatment level had occurred in 84 per cent of

tolerated, but because of the inconvenience and lack of need for its use intravenously, such preparations are not commercially available.

For topical use in the eye, suspensions of cortisone acetate, 25 mg per 1 ml., and 5 mg. per 1 ml., have been prepared. Also, an ophthalmic ointment of cortisone acetate, 1.5 per cent, is available.

Hydrocortisone.—This steroid has been prepared as a suspension of microcrystals of the acetate salt, 1 ml. containing 25 mg When used intramuscularly, this suspension has an action similar to that of cortisone acetate It may also be injected directly into body cavities, and is commonly used intra-articularly for a local action, as will be discussed later (Chapter 18).

Tablets of hydrocortisone acetate have been studied, but they possess no advantage over cortisone acetate. However, hydrocortisone, the free alcohol, has been considered of sufficient superiority (under some conditions) over cortisone acetate that it has been prepared in tablets containing 20 mg. Potency of hydrocortisone has been reported to be substantially 50 [illegible]

sn [illegible]

tr [illegible]

sterile water or saline shortly before its use intramuscularly. A wide range of concentration could be made, for this hormone is quite soluble. The action of this preparation is rapid and usually decreased to a small magnitude within six to eight hours after injection, so that its use was most effective and efficient if it were injected at six-hour intervals. This schedule of administration soon becomes very strenuous for the patient as well as the nurse or physician, and its usefulness is thereby limited to such an extent that there have been many attempts to make a preparation which will act as a depot from which only slow and prolonged absorption of the injected hormone will result. Different forms of this delayed-acting or reservoir corticotropin are now available and indications are that injections given at intervals of twelve to twenty-four hours are quite practical. Some of these long-acting preparations of corticotropin, although not greatly irritating, may cause induration at the injection site, which may liquefy and result in troublesome abscesses. Further research has led to the development of long-acting preparations that are better tolerated and which, under many circumstances, have considerable advantage over the crystalline preparation

There are now some stable solutions of crystalline corticotropin marketed in this form for clinical use.

Crystalline corticotropin may be given intravenously, mixed with a large volume (usually 1 liter) of physiologic, sterile saline or glucose solution Given intravenously slowly over a period of six or eight hours, stimulation of the adrenals is as great from 20 mg. of corticotropin as is obtained from four or five times this amount given intramuscularly in divided doses at six-hour intervals through the day. Hence, for producing a quick effect corticotropin used intravenously has a distinct advantage, but for prolonged use this manner of administration is obviously impractical.

Attempts have been made to use corticotropin orally, but due to its protein nature it is readily and quickly hydrolyzed in the alimentary tract so that no satisfactory hormone action results. Because its effects

TABLE 16

COMPLICATIONS OCCURRING DURING MAINTENANCE CORTISONE IN 44 PATIENTS

Type	*Incidence*					
	Degree					
	+	++	+++	++++	*Total*	*Per cent*
Round face	10	2	1		13	30
Obesity	6	2			8	18
Hirsutism	4				4	9
Acne	2				2	4
Edema	14	6	2		22	50
Potassium decrease	4					9
Diabetes		1		1	2	4
Irregular menstruation	8				8	32
Menopausal symptoms	5				5	20
Jitteriness, insomnia	9	2			11	25
Excitement	2				2	4
Depression			1		1	2
Tachycardia	12	2	2		16	36
B.P elevation	11	3			14	32
Telangiectasis	2				2	4
Purpura	3	2			5	11
Hemorrhage (G-I)				2	2	4
Thrombosis (leg)	2				2	4
Thrombosis (coronary)					0	0
Cardiac decompensation					0	0

Complications predominated in females
Some abnormalities listed may not be due to cortisone

TABLE 17

POST-CORTISONE EVENTS IN 24 CASES

	Number	*Per cent*
Continued improved (50 to 75 per cent), 60 to 190 days	4	16
Relapsed to pre-treatment condition		
Within 10 days	6	25 } 84
10 to 20 days	8	33
20 to 30 days	4	16
30 to 60 days	2	8
Total	24	

Includes 2 cases of early disease (6 and 8 months' duration)
Includes many cases treated 200 to 370 days

Adrenocortical insufficiency—temporary	10	40
"*Withdrawal syndrome*"	5 (4 females)	21
Fracture of the hip	2	8
Edema	3	12
Difficult readjustment to worsened disease	4	16

result only from stimulation of the adrenal glands, topical or regional use of corticotropin is ineffectual

TABLE 15

CHIEF CLINICAL USES OF CORTISONE AND CORTICOTROPIN

Very Beneficial	*Beneficial—But Variable Effects*	*Limited Benefit*
Endocrinopathies	Bronchial asthma	Leukemia
Adrenal cortical insufficiency	Hay fever	Hodgkin's disease
Panhypopituitarism	Acute gouty arthritis	Lymphosarcoma
Hypoglycemia, idiopathic	Bursitis	Multiple myeloma
	Dermatomyositis	Chronic atopic dermatitis
Rheumatic Diseases	Erythema nodosum	Scleroderma
Acute rheumatic fever	Ulcerative colitis	Acute glomerulo-nephritis
Rheumatoid arthritis	Burns	
Disseminated lupus erythematosus	Sarcoidosis	
Periarteritis nodosa	Berylliosis	
	Silicosis	
Hypersensitivity Diseases	Nephrotic syndrome	
Serum sickness	Pemphigus	
Drug sensitivity reactions		
Contact dermatitis		
Exfoliative dermatitis		
Angioneurotic edema		
Erythroblastosis fetalis		
Acute Inflammatory Eye Diseases		
Iritis		
Keratitis		
Uveitis		
Choroiditis, etc		

CLINICAL CONSIDERATIONS

When the clinical effects of cortisone and corticotropin were first announced, Hench and his collaborators emphasized that *they were reporting not on a new treatment, but a physiological experiment, and they cautioned against an immediate attempt to use these substances for treatment* They considered them important research tools, as indeed they are However, as supplies became more abundant, it was only natural that careful attempts were made to use these hormones in the practical management of patients From the multitudinous literature discussing clinical trials, the uses of cortisone and corticotropin for various diseases can be grouped as shown in Table 15 Certain rheumatic diseases are seen to occupy prominent places in this review. It should be noted that the forms of rheumatism [illegible]

[illegible] of the inflamed joints [illegible] in the joints

the patients! In many instances relapse started when the daily dose was reduced below 50 mg It is considered important that even the patients with early disease relapsed promptly after discontinuance of cortisone Also, relapse occurred as often in those patients treated for 200 to 370 days as in those treated for 100 to 200 days. As suspected, there was laboratory evidence of suppressed adrenal function in many patients, but this recovered within two to six weeks.

A very distressing illness which we call "post-cortisone withdrawal syndrome" occurred in 21 per cent of patients. Again, note that 4 of these patients (80 per cent) were females. This syndrome is characterized by

everything The syndrome always subsided in two to eight weeks, but some patients still felt more ill then, than before treatment

Whenever possible it was found advisable to discontinue cortisone gradually so that the metabolic, endocrinological and psychological adjustments were made more easily Often patients with no complication during treatment found it very difficult to adjust psychologically to their worsened state after the "holiday" affected by cortisone.

Based upon these experiences, which are similar to those reported by Boland[4] and by Ward, Slocumb *et al*[47] this type of *maintenance cortisone treatment appears to be valuable for some, but not practical for all patients with rheumatoid arthritis Hence, it seems reasonable that such treatment should not be considered as satisfactory routinely, for patients with rheumatoid arthritis* but should instead be carefully selected for special purposes, such as a rapidly worsening disease, or to facilitate physical treatment aimed toward rehabilitation

USE IN OTHER CHRONIC RHEUMATIC DISEASES

Rheumatoid Variants.—Rheumatoid spondylitis (Marie-Strumpell dis-

clearly that the dose of cortisone is governed not by the size or age of the patient, but by the severity of the disease. Usually the child's requirement approaches or equals the dose needed for adults, and when so used in children, more complications occur than are observed in adults receiving similar doses of cortisone

Osteoarthritis.—Boots[5] has reported relief of pain, easier movement and

such patients chiefly, or only, by [illegible]
strength and by giving a general "
results primarily from degeneratio
cortisone or corticotropin cannot result through its anti-inflammatory action, and because the anatomic joint changes are irreversible it appears

and in the nonarticular connective tissue lessens, often disappears. Joint swelling, tenderness and heat progressively decrease. The time required for these changes to be completed varies from a few days in acute diseases to several weeks in more chronic or more severe forms of rheumatism. If treated with sufficient amounts of cortisone or corticotropin early in the illness, complete disappearance of all physical signs of inf

all symptoms frequently subside. Signs and symptoms of accompanying nonarticular inflammations—bursitis, tenosynovitis, nodules and other nonarticular features—improve or vanish concurrently with improvement in the joint synovitis. If destructive changes affecting cartilage or bone have occurred, as are frequently encountered in chronic rheumatoid arthritis, these anatomic abnormalities do not improve. Thinning of articular cartilage, cystic changes in bone, osteoporosis, exostoses, calcification of ligaments, intra-articular ankylosis (fibrous or bony) and structural joint deformities all remain unchanged.

While rheumatic abnormalities are improving there is comparable improvement in systemic manifestations of illness. If present, fever subsides, appetite improves and may become ravenous, so that there is a remarkable increase in consumption of food, weight gain, strength, and general sense of well-being. Euphoria is frequently prominent.

The laboratory abnormalities which characterize the illness change toward or to normal. Anemia improves or is abolished, plasma protein composition changes toward normal—the globulin decreases and albumen increases. Almost invariably a very significant reduction occurs in the sedimentation rate of erythrocytes; the degree of reduction usually parallels the amount of clinical improvement. Since the anatomic abnormalities at the joints do not change, no improvement can be expected to appear in roentgenograms of the joints.

The three predominant attributes which characterize cortisone and corticotropin are: (1) dependability of action; (2) speed of effect; and (3) the high order of benefit obtained. The degree of anti-rheumatic effect, like the other effects of these hormones, is directly related to the quantity of the biologically effective hormone administered; the action of these hormones can be finely regulated by adjustment of the dose so that the activity of disease can be decreased or allowed to increase at will. The anti-rheumatic effect continues so long as cortisone or corticotropin administration is continued, but in ... atic illnesses th ... t acute or self-limited, relapse usually o ... ssation of th ... The speed and degree of relapse var ... tably under ... reum-stances.

Manner of Action.—The way i ... isone and ... pro-
duce this ... e anti-rheu ... is not k ... ough
Dobriner[8] h ... at certain ... ids and ... nti-
ties of natu ... are excret ... toid art ... in
that some a ... adrenal ... xist in
form of rheu ... is no cl ... t any
is caused by ... adrenal ... efi
The administ ... e to a
deficiency of t ... d, t

that *these hormones have a very limited place, if any, in the management of this form of rheumatism.* (*See* Chapter 40.)

Fibrositis.—This common form of non-articular rheumatism is usually

cortisone or corticotropin, but had complete relapse promptly after the hormone was discontinued. *Because of the mild nature of this rheumatic disorder and the tendency to chronicity, it seems unwise to use such powerful agents as cortisone and corticotropin for treatment.* (*See* Chapter 35.)

For painful, stiff ("frozen") shoulder, and for shoulder-hand syndrome several investigators have reported very good relief of pain and liberation of motion with use of rather large doses of cortisone or corticotropin for a few weeks while intensive physical treatment and exercises are employed If acute bursitis or tendonitis about the shoulder subsides, but leaves stiffness of the shoulder which is not relieved by other measures, it seems justified to use hormone therapy to accomplish the best results from physical treatment. (*See* Chapter 54.)

Erythema Nodosum.—Erythema nodosum is oftentimes a rather mild but troublesome febrile disease, but it may be severe and confine the patient to bed for many weeks. In these more severe cases, unrelieved by more conservative measures of treatment, cortisone or corticotropin has controlled the disease promptly and completely. As in patients with rheumatic fever, these patients with erythema nodosum usually need treatment for only a few weeks, after which the disease may remain quiescent when the hormones are withdrawn. Should recurrences ensue, re-institution of treatment is usually effective.

Diffuse Collagen Diseases.—This group includes disseminated lupus erythematosus, scleroderma, dermatomyositis, and polyarteritis. All are chronic, distressing, frequently disabling illnesses which have a grave prognosis and a very high mortality rate. *In early or acute stages they may be benefited by cortisone or corticotropin.* (*See* Chapter 36.)

Disseminated Lupus Erythematosus is often greatly benefited by proper administration of these hormones; indeed, cortisone and corticotropin have been the only agents that have rather consistently exerted significant control over the progressive decline that usually characterizes this grave illness. Occasionally long remissions have followed the vigorous use of these hormones in the early stage of illness, but usually attempts to discontinue hormone treatment have been followed by relapse so that commonly cortisone or corticotropin need to be continued for an indefinitely prolonged time. Although all or most of the numerous manifestations of the disease can usually be suppressed, studies have repeatedly shown persistence of the characteristic pathologic "lupus erythematosus cells" in circulating blood or in the bone marrow, indicating that the pathologic process is not arrested. Many patients, however, are kept alive and comfortably active for years by prolonged treatment with cortisone or corticotropin; failure of one of the vital organs results in death. Since this disease is nearly universally fatal if untreated, it seems not only justified but perhaps essential to use one of these hormones as skillfully as possible for the most effective control of this disease. (*See also* Chapter 36.)

Usually large daily doses, 100 mg or more of cortisone, or 50 mg. or more of corticotropin, are required for long periods in order to suppress this illness In some cases as much as 1000 or 2000 mg. of cortisone have been given daily to patients in acute crisis[17]

Because of the common requirements for large doses and the frequent severe cardiac, renal or pleural involvement, complications are more common with the use of these hormones for this illness Special care must be taken to prevent troublesome edema, congestive heart failure, potassium deficiency and other serious "toxic" effects. But many less serious, undesirable side-effects of these hormones frequently must be tolerated but minimized in order to use these hormones for their possible life-prolonging

In some patients treated for long periods, death has occurred from cardiac or renal failure, and autopsies have shown extensive arterial scars occluding the lumen of vessels, where inflammatory lesions had existed. Such consequences of hormone treatment are unfortunate, but it is likely that the disease, untreated, would have resulted fatally from progressive inflammatory lesions

plished which e

hormone, whi s

occur

Benefit of *limited value* has been observed in patients with *scleroderma*. The writer has seen slow but definite softening and thinning of affected skin to occur in early cases treated with moderate doses of cortisone or corticotropin for many months. In many advanced cases even large doses have failed to effect practical benefit. The place of these hormones in the management of this poorly understood disease, will need longer study to clarify (*See* Chapter 37)

Treatment of *dermatomyositis* has produced variable results. In some patients the disease has been kept suppressed significantly for long periods, in other instances little benefit has been obtained Trial is quite justified in any instance in which the disease is progressing in spite of conservative therapy.

LIMITATIONS OF PRACTICAL USEFULNESS

This rather brief review of the use of cortisone and corticotropin in various

may

Some

which materially limit their practical usefulness in management in some patients High hopes for widespread applicability were entertained early in the period of study of these hormones, with increasing experience, their limitations have become well defined, and are seen to markedly restrict their usefulness to the degree that the routine administration is unwarranted.

In acute rheumatic disorders which require the administration of cortisone or corticotropin for only short periods to effect important clinical benefit, there is little or nothing to interfere with their general use. The problems of administration and complications that may occur are generally

manner of action of these potent hormones has not yet been elucidated. The effects are temporary, suppressive but not curative, and there is no evidence that the ultimate course of a *chronic* rheumatic disease is favorably altered by these hormones, as they have been employed. There are potential dangers involved in using these materials, great care and definite precautions must be taken. These hormones can be put to practical use in treatment of patients, but the significant potential complications and limitations in their usefulness have thus far kept these agents from being *routinely* used to treat inflammatory rheumatic diseases Indeed, the use of these hormones has become quite highly selective and for the purpose of realizing definite objectives

The physician using these substances should have full realization of their limitations and the problems as well as the potential benefits involved. Troubles must be expected to occur in some patients, and the physician should be prepared to meet them. One should consider not only what may happen *during* the use of these hormones, but also what might occur *after* their use.

as

us

held, gloom and despair are not warranted Instead, with the impetus

investigations to solve the problems which remain, so as to provide the means of prevention or more successful treatment of these prevalent and serious rheumatic diseases Never before has the hope for success been so justified and the future of rheumatology been so bright!

BIBLIOGRAPHY

1 Aldersberg, D. *et al* J Clin Invest, 30, 626, 1951
2 Barnes, A R. Circulation, 3, 770, 1951
3 ——— Am J Dis Child, 82, 397, 1951
4 Boland, E W, and Headley, N. E J Am Med Assoc, 147, 1538, 1951
5 ——— J Am Med Assoc, 148, 981, 1952
6 Boots, R. H, Yen, L C, McMarrow, K J, and Ragan, C J Am Med Assoc, 147, 549, 1951
7 Conn, J W, Louis, L H, and Fajans, S S Science, 113, 713, 1951
8 Dobriner, K Address, Annual Meeting American Rheumatism Association,

1951
15 Gray, S J, Spiro, H M, and Reifenstein, R. W Bull New England Med Centre, 12, 160, 1950
16 Gray, S J, Reifenstein, R. W, Connally, E. P *et al* Gastroenterology, 16, 687, 1950
17 Haserick, J R., Corcoran, A C, and Dustan, H P. J Am Med Assoc, 146, 643, 1951.

encountered infrequently or to a mild degree in such acute illnesses. *The important complications and limitations of these hormones, in the field of rheumatism, are met most often in the management of chronic illnesses.* These problems are due to the following:

1. Although possessing remarkable power for correcting metabolic abnormalities and reversing the pathologic physiology of some diseases, cortisone and corticotropin have not the power to eliminate the pathogenic factors, hence *they cannot be expected to cure* any disease.
2. The value of cortisone administered directly or secreted under stimulation of corticotropin lasts only during administration of the hormones, thus there is an important *temporal limitation.*
3. Cortisone does not repair damaged tissue (articular cartilage, bone) or eliminate scarred connective tissue, hence these hormones *cannot be expected to restore deformed arthritic joints, or cardiac valves.*
4. The hormones have so *many physiologic actions which cannot be divorced from their anti-rheumatic effect,* that in attempting to obtain the latter, many undesirable metabolic and hormonal effects may result to the degree that they may exceed the desired action
5. *Corticotropin must be administered by injection*; this presents a practical problem when prolonged use is required in ambulatory patients.

INDICATIONS FOR USE OF CORTISONE AND CORTICOTROPIN

In discussing any treatment one naturally would desire to state precisely the indications, and the conditions under which it should not be employed. Unfortunately, at the time of this writing, in the case of cortisone and corticotropin, this is not possible. Indeed, the opinions of various investigators do not entirely agree, and until larger experience over longer time is critically evaluated, dogmatic statements and unqualified recommendations for the clinical use of these hormones cannot be made.

From discussions in foregoing sections of this chapter, it is apparent that any patient with an *acute inflammatory* rheumatic disease may be considered a candidate for the use of cortisone or corticotropin, especially if the disease appears to be advancing and other treatments have been [illegible] However, the relative merits of these hormones,

[illegible]

able knowledge.

Among patients with *chronic* rheumatic disorders it is even more difficult to select candidates for treatment. In general, the non-inflammatory illnesses (osteoarthritis, post-traumatic arthritis), will benefit so *little,* that realizing the problems attendant thereto, prolonged use of these hormones seems unjustified. Any patient with rheumatoid arthritis may be expected to receive significant benefit from cortisone or corticotropin; the important [illegible]

question. Many patients with rheumatoid arthritis have such a mild form of the disease that conservative measures of treatment will give excellent

18 HENCH, P S, KENDALL, E C., SLOCUMB, C. H., and POLLEY, H F.: Proc. Staff Meet., Mayo Clinic, 24, 181, 1949
19. HENCH, P. S, SLOCUMB, C. H *et al* · J. Am. Med Assoc., 144, 1327, 1950.
20. HENCH, P. S.: Personal Communication.
21. HENDERSON, E. *et al* J. Clin Endocrinol, 10, 800, 1950.
22. HILL, S R., JR, REISS, R. S, FORSHAM, P. H, and THORN, G. W · J. Clin Endocrinol, 10, 1375, 1950.
23. HOLLANDER, J. L.: Personal Communication.
24. HOLLANDER, J. L, BROWN, E. M, JR, JESSAR, R. A, and BROWN, C. Y.· J Am. Med. Assoc., 147, 1629, 1951
25. INGLE, D J · Ann New York Acad. Sc, 50, 576, 1949
26. KENDALL, E. C.: Lancet, 2, 586, 1951
27. MARGOLIS, H M, and CAPLAN, P S · J. Am. Med Assoc, 145, 382, 1951.
28. MASSELL, B F, and WARREN, J. E.· J Am. Med Assoc., 144, 1335, 1950.
29. MCEWEN, C. *et al*. Bull N Y Acad Med., 26, 212, 1950
30 PATTERSON, M, FREYBERG, R. H, and SEIGEL, H · Unpublished Data
31. PEARSON, O H., ELIEL, L P, and HOLLANDER, V P.: J Clin Invest, 30, 665, 1951.
32. PERERA, G A: Proc. Soc Exper. Biol. and Med, 76, 583, 1951.
33. POLLEY, H F, and MASON, H. L · J. Am Med. Assoc, 143, 1474, 1950
34 RAGAN, C, HAWSE, E L., PLOTZ, C. M. *et al* Proc. Soc. Exper Biol. and Med, 72, 718, 1949
35. RAGAN, C. Proc Conf on Effects of Cortisone, Merck & Co, Inc., Rahway, N J., 54, 1951.
36. RICH, A. R. *et al* Bull. Johns Hopkins Hospital, 88, 189, 1951.
37. ROSE, B, PACE, J A P, PUMP, K, and STANFORD, R. L: Canadian M. A J, 62, 6, 1950.
38. SPRAGUE, R G *et al* Arch. Int. Med., 85, 199, 1950.
39. SPRAGUE, R. G. Am Jour. Med, 10, 567, 1951.
40. STEVENSON, C, ZUCKNER, J. and FREYBERG, R. H · Ann Rheum. Dis, 11, 112, 1952
41. THORN, G W *et al* J. Clin Investigations, 19, 813, 1940.
42. THORN, G W, FORSHAM, P H, PRUNTY, F. T G, and HILLS, A G J. Clin. Endocrinol, 8, 15, 1948.
43 ——— J Am. Med Assoc, 137, 1005, 1948
44 THORN, G W New England J Med, 242, 783 and 824, 1950
45. ———: Personal Communication
46 TRAEGER, C. H, BATTERMAN, R, and WAINERDI, H.: Personal Communication
47. WARD, L. E., SLOCUMB, C. H *et al* Proc. Staff Meet., Mayo Clinic, 26, 361, 1951.
48. WEDGWOOD, R J, HAWN, V, and JANEWAY, C. A: Proc Second Clinical ACTH Conf, Philadelphia, Pa, Blakiston Co, 1, 108, 1951.
49. WIESEL, L L, BARRITT, A S, and STUMPE, W. M.: Am. J. Med Sci, 222, 243, 1951.

results, and special treatments will not be needed. Another group of

and justified to employ cortisone or corticotropin as wisely as possible to suppress the disease and hope to prevent crippling. Another circumstance in which these hormones may be very beneficial exists in those patients who have been left disabled from previously active arthritis in whom the skillful use of cortisone or corticotropin combined with good physical treatment may be distinctly helpful to accomplish quicker and greater restoration of joint and muscle function and thus materially aid in the patient's rehabilitation.

In some of the collagen diseases with grave prognosis (such as diffuse lupus erythematosus) use of cortisone or corticotropin seems entirely justified even though troublesome complications may occur, for these illnesses may be suppressed and life prolonged only by their use

CONTRAINDICATIONS

Just as in the consideration of their usefulness, one cannot list many absolute contraindications to the use of cortisone or corticotropin, most conditions present "relative contraindications" which must be weighed

lem During *acute psychoses*, *active tuberculosis* and *syphilis*, and in patients with an *active peptic ulcer*, these hormones *should not be used*. There are many situations that should be considered relative contraindications so that great care must be exercised if use of these hormones is strongly desired The history of important psychosis or major emotional or mental disturbances should make one very hesitant to use cortisone or corticotropin, most psychoses that have occurred as complications during treatment with these substances have been in patients with such a background Recently healed peptic ulcers may be reactivated; when long periods of freedom from ulcer distress have intervened (presumably healed completely) these hormones may not initiate recurrence. Similar considerations are justified for long-arrested tuberculosis. Severe *hypertension* may be increased to result in undesirable complications, but in patients with only small increases in blood pressure, the hormones may not provoke trouble, but extreme care must be exercised in such situations. Similarly, *severe diabetes* may prevent the use of cortisone and corticotropin, but in mild diabetic states, cautious use may be satisfactory In renal insufficiency or high grade congestive cardiac failure these hormones may be dangerous due to unfavorable alterations in the metabolism of inorganic elements In mild cardiac or renal disease these troubles may be prevented In the presence of marked osteoporosis, prolonged use of cortisone and corticotropin should be avoided in order to prevent further softening of the bone

CHAPTER 18

INTRA-ARTICULAR INJECTION OF HYDROCORTISONE*

By JOSEPH LEE HOLLANDER, M.D.

To the time of this writing, we[1 6,7] have injected hydrocortisone acetate into the arthritic joints of 723 patients a total of 7,352 times. In our hands, and in those of other rheumatologists[13 14 16,20] who have tried this method, it has proved a practical aid in the local treatment of many types of arthritic inflammation. If the arthritis is limited to, or is particularly active in, only one or a few peripheral joints, it can be suppressed by repeated injections of the hormone into each, without the dangers attending *systemic* administration of cortisone or hydrocortisone. The technics for use, indications and *contraindications*, *limitations*, and *precautions* for this method of local hormone therapy will be set forth in this chapter. It is specified at the outset that this method is only an *adjunct* in arthritis treatment, and *must be coordinated with the general plan of therapy.*

INTRODUCTION

The idea of injecting a medication directly into the joint space is not new For many years arthritic patients have asked their physician why he could not give something "directly into the joint that hurts" The local application of heat, and the use of liniments have thus appealed to the patient because he sees and feels something is being done directly to the painful spot.

Injection of liquid petrolatum into the joint space in an effort to provide "better lubrication" was tried years ago, but failed because the oil was not miscible with joint fluid, and was soon pocketed off in one or more of the pouches of the synovial sac. Lipiodol, injected into the joint space originally for visualization of the synovial sac by roentgenogram, was later tried for treatment of arthritis, but also quickly became localized in a synovial pouch, often walled off by fibrin and the pannus of the rheumatoid arthritic process

A solution of procaine hydrochloride has been frequently injected into the synovial cavity of inflamed joints by the physician treating a chronic synovitis produced by trauma to the joint. Such injections have been [illegible]

has been injected directly into the joints.

* Some of the text on technic of injection, and figures 97–104, were originally prepared by the author for a pamphlet to be issued to physicians by Merck and Co, Inc. They are reproduced here by special agreement with Merck and Co, Inc who sponsored this part of the work

which may result in fracture. *Bleeding tendencies* may make more hazardous the use of these hormones; if they are employed in such conditions sharp watch should be maintained to recognize and satisfactorily manage hemorrhage. During *acute infections* use of cortisone or corticotropin should be discontinued in order to favor localization of the inflammation and allow satisfactory cure. No difficulty in healing of operative or accidental wounds has been experienced among the writer's patients receiving these hormones in usual therapeutic doses.

GENERAL PRECAUTIONS

The most important safeguard for the patient receiving cortisone or corticotropin is a thorough understanding of the physiological effects of these agents by the physician. The patient should be kept under close surveillance throughout the treatment and routine clinical checks should be made regarding nervous tension, excitability, insomnia, personality changes, weight, pulse, blood pressure, edema and menstrual disorders. Weekly or bi-weekly urine analyses, erythrocyte sedimentation rate and serum potassium determinations are advisable, and if troubles occur as to so indicate, other blood chemistry studies, electrocardiogram, electroencephalogram or basal metabolic rate determination may be helpful.

PREVENTION OF COMPLICATIONS

Obesity.—Avoidance of excess hormone administration and use of a diet adequate in essentials but low in calorie value will usually prevent serious trouble from obesity. However, fatty jowls, round face and deposits of fat at the base of the neck posteriorly ("buffalo-hump"), above the clavicles and about the hips, especially in females, may occur even with these precautions Androgenic effects resulting in hirsutism and acne may also occur, but when the dose of hormones is not high, these are usually only nuisance troubles which do not prevent continuation of treatment.

Edema.—In many patients edema does not occur when the hormone is [illegible]

containing appreciable amounts of sodium (such as sodium salicylate, sodium bicarbonate and infusions of physiologic saline) should be avoided Ammonium chloride and mercurial diuretics may be needed at intervals in order that the hormone may be continued without trouble from edema. When diuretics are used special attention is required to prevent deficiency of potassium, for this element is excreted in larger amounts as a result of the action of the diuretic. In patients with heart disease, hypertension or renal disease, edema tends to develop more often and to a greater degree; these patients should be especially carefully checked in regard to edema during use of these hormones.

Potassium Deficiency.—The excretion of large amounts of potassium, leaving a tissue deficiency, happens from use of corticotropin or cortisone much less than expected. For this reason it seems unnecessary routinely

18. Hench, P. S., Kendall, E. C., Slocumb, C. H., and Polley, H. F.: Proc Staff Meet., Mayo Clinic, 24, 181, 1949
19. Hench, P. S., Slocumb, C. H. *et al.*: J. Am. Med Assoc., 144, 1327, 1950.
20. Hench, P. S.: Personal Communication.
21. Henderson, E. *et al*: J. Clin Endocrinol., 10, 800, 1950.
22. Hill, S. R., Jr., Reiss, R. S., Forsham, P. H., and Thorn, G. W.: J. Clin. Endocrinol., 10, 1375, 1950
23. Hollander, J. L.: Personal Communication.
24. Hollander, J. L., Brown, E. M., Jr., Jessar, R. A., and Brown, C. Y.: J. Am. Med Assoc., 147, 1629, 1951
25. Ingle, D. J.: Ann New York Acad. Sc., 50, 576, 1949
26. Kendall, E. C.: Lancet, 2, 586, 1951
27. Margolis, H. M., and Caplan, P. S.: J. Am Med Assoc., 145, 382, 1951.
28. Massell, B. F., and Warren, J. E.: *J. Am. Med. Assoc.*, *144*, *1335*, *1950*
29. McEwen, C. *et al*: Bull N. Y. Acad Med., 26, 212, 1950.
30. Patterson, M., Freyberg, R. H., and Seigel, H.: Unpublished Data
31. Pearson, O. H., Eliel, L. P., and Hollander, V. P.: J. Clin Invest., 30, 665, 1951.
32. Perera, G. A.: Proc Soc. Exper Biol and Med., 76, 583, 1951.
33. Polley, H. F., and Mason, H. L.: J. Am Med Assoc., 143, 1474, 1950
34. Ragan, C., Hawse, E. L., Plotz, C. M. *et al.*: Proc Soc Exper. Biol and Med., 72, 718, 1949
35. Ragan, C.: Proc Conf on Effects of Cortisone, Merck & Co., Inc., Rahway, N. J., 54, 1951.
36. Rich, A. R. *et al*: Bull Johns Hopkins Hospital, 88, 189, 1951.
37. Rose, B., Pace, J. A. P., Pump, K., and Stanford, R. L.: Canadian M. A. J., 62, 6, 1950
38. Sprague, R. G. *et al*: Arch. Int Med., 85, 199, 1950.
39. Sprague, R. G.: Am Jour Med., 10, 567, 1951.
40. Stevenson, C., Zuckner, J. and Freyberg, R. H.: Ann Rheum. Dis., 11, 112, 1952
41. Thorn, G. W. *et al*: J. Clin. Investigations, 19, 813, 1940.
42. Thorn, G. W., Forsham, P. H., Prunty, F. T. G., and Hills, A. G.: J. Clin Endocrinol., 8, 15, 1948
43. — [illegible]
44. T[illegible] 0
45. — [illegible]
46. Traeger, C. H., Batterman, R., and Wainerdi, H.: Personal Communication.
47. Ward, L. E., Slocumb, C. H. *et al*: Proc. Staff Meet., Mayo Clinic, 26, 361, 1951.
48. Wedgwood, R. J., Hawn, V., and Janeway, C. A.: Proc Second Clinical ACTH Conf., Philadelphia, Pa., Blakiston Co., 1, 108, 1951
49. Wiesel, L. L., Barritt, A. S., and Stumpe, W. M.: Am J Med Sci., 222, 243, 1951.

to administer potassium chloride as prophylaxis, especially since the potassium salt may provoke nausea and indigestion, or even cause excess tissue potassium if given when not needed. A wise course is to measure the serum

Should potassium deficiency occur, potassium chloride is a very effective supplement; one or two grams daily usually corrects a deficiency and maintains normal tissue concentration; seldom is more than this needed. Determination of serum potassium content is the best guide for the appropriate dose of potassium.

In patients who are febrile and gravely ill, as are some with acute rheumatic fever or disseminated lupus erythematosus, loss of potassium may produce alarming changes. These patients should be carefully observed from this standpoint It is advisable to administer potassium chloride prophylactically if a mercurial diuretic is given.

Disturbance of Carbohydrate Metabolism.—Usual symptoms of diabetes mellitus indicate the development of decreased carbohydrate combustion which can be readily confirmed by urinalysis and blood sugar determinations Routine urinalysis for sugar at frequent intervals is recommended in order to recognize such disturbance early in its development, for usually it is necessary or wise to discontinue administration of the hormone in this event. Ketosis may not develop even with severe decrease in utilization of sugar If faulty carbohydrate metabolism develops it is usually obvious within the first weeks of treatment, seldom does such trouble occur after the early weeks of treatment, even with prolonged use of hormones

Osteoporosis.—It seems reasonable to expect measures usually helpful

ministration. This treatment is only now being studied; results should be forthcoming soon

Ovarian Suppression.—Menstrual irregularities, hypomenorrhea or complete amenorrhea may occur, particularly in female patients in the premenstrual age These menstrual changes and the neurovascular menopause disturbances that follow are always a source of worry to the patient. They always disappear, if not during, then after treatment They can usually be prevented or significantly reduced by adequate supplements of estradiol *benzoate orally, or by the injection of appropriate doses of estrogens* intramuscularly.

Nervous and Mental Changes—Most helpful to prevent nervous and

these hormones In any patient, however, important psychotic disturbances can usually be prevented if special care is used early in the administration of these hormones A feeling of "being driven," "nervous tension," unusual euphoria, or carefreeness similar to that which results from "too

In our own laboratory, we have recently determined that the hormone rapidly disappears from suspension in the synovial fluid after injection, and seems to be taken up by the cells of the joint fluid and by the lining cells of the synovial membrane.[8] The length of time it is stored or utilized in these cells has not been determined up to the time of this writing, but seems quite variable—depending on the individual, the type of arthritis, and the degree of activity of the process.

We have made intensive comparative studies[1,7] to see whether other steroid compounds would have a similar anti-inflammatory effect. Apparently cortisone (free alcohol), cortisone tricarballylate, and cortisone propionate have no more effective local action in the joints than cortisone acetate. The free alcohol of hydrocortisone, however, appears to be as effective as the hydrocortisone acetate usually used. Apparently solubility alone is not a primary factor in effectiveness.

CLINICAL INDICATIONS FOR INTRA-ARTICULAR HYDROCORTISONE INJECTION

After nearly two years of trial, we have concluded[1] that intra-articular hydrocortisone is clinically useful for local palliative therapy in the following conditions:

1. When one or only a few peripheral joints are inflamed or painful from rheumatoid arthritis, osteoarthritis, traumatic arthritis, acute or chronic gouty arthritis, or intermittent hydrarthrosis. (It may also be used with benefit in acute inflammation of the subdeltoid bursa, by injection directly into the bursal sac, but is rarely helpful in chronic or calcific bursitis of the shoulder.) It should *never* be injected into a joint in the presence of *infection*, since the hormone appears to decrease resistance to infection in the tissues. Hydrocortisone *must not be injected into a joint until a diagnosis has been established*, and specific infectious arthritis ruled out
2. In rheumatoid or osteoarthritis particularly active in a few peripheral joints, even in the presence of more widespread low grade joint involvement, the employment of this method as an adjunct to suppress the activity in these few worst joints is helpful.
3. In rheumatoid arthritis, intra-articular hydrocortisone can be used to suppress activity in the most severely involved joints until the beneficial effects of gold therapy, or other general measures, become established
4. In rheumatoid arthritis cases in which gold therapy, or systemic ACTH or cortisone therapy is contraindicated because of cardiac failure, renal impairment, hypertension, diabetes, or other reason, the injection of hydrocortisone into the most active joints may be useful
5. When systemic cortisone, hydrocortisone or ACTH therapy controls the rheumatoid arthritic activity in all but a few "resistant" joints, intra-articular injection into such joints can accomplish a more complete suppression of the process on a smaller systemic (and total) dose of hormone.

which may result in fracture. *Bleeding tendencies* may make more hazardous the use of these hormones; if they are employed in such conditions sharp watch should be maintained to recognize and satisfactorily manage hemorrhage. During *acute infections* use of cortisone or corticotropin should be discontinued in order to favor localization of the inflammation and allow satisfactory cure. No difficulty in healing of operative or accidental wounds has been experienced among the writer's patients receiving these hormones in usual therapeutic doses.

GENERAL PRECAUTIONS

The most important safeguard for the patient receiving cortisone or corticotropin is a thorough understanding of the physiological effects of these agents by the physician The patient should be kept under close surveillance throughout the treatment and routine clinical checks should be made regarding nervous tension, excitability, insomnia, personality changes, weight, pulse, blood pressure, edema and menstrual disorders Weekly or bi-weekly urine analyses, erythrocyte sedimentation rate and serum potassium determinations are advisable, and if troubles occur as to so indicate, other blood chemistry studies, electrocardiogram, electroencephalogram or basal metabolic rate determination may be helpful

PREVENTION OF COMPLICATIONS

Obesity.—Avoidance of excess hormone administration and use of a diet adequate in essentials but low in calorie value will usually prevent serious trouble from obesity. However, fatty jowls, round face and deposits of fat at the base of the neck posteriorly ("buffalo-hump"), above the clavicles and about the hips, especially in females, may occur even with these precautions. Androgenic effects resulting in hirsutism and acne may also occur, but when the dose of hormones is not high, these are usually only nuisance troubles which do not prevent continuation of treatment.

[illegible]

in order that the hormone may be continued without trouble from edema [illegible] to prevent deficiency [illegible] ounts as a result of [illegible] se, hypertension or renal disease, edema tends to develop more often and to a greater degree, these patients should be especially carefully checked in regard to edema during use of these hormones.

Potassium Deficiency.—The excretion of large amounts of potassium, leaving a tissue deficiency, happens from use of corticotropin or cortisone much less than expected. For this reason it seems unnecessary routinely

Intra-articular injections of lactic acid have been recommended to "*stimulate natural local tissue reactions* and evoke processes of repair in traumatized joints."[10,18,19] This was carried out by a few British workers, and has not been generally corroborated nor accepted by most rheumatologists

Shortly after the discovery of the anti-rheumatic effect of cortisone, Thorn[17] injected 10 mg. of compound F (hydrocortisone) into the knee joint of a patient with rheumatoid arthritis The next day the injected knee was greatly improved, but there was also a coincidental general improvement in the patient's condition. He assumed the patient was very sensitive to the systemic action of the hormone, and did no further experiments along this line.

We[6,7] attempted injections of 25 mg. or more of cortisone acetate into inflamed knees of rheumatoid arthritic patients. In less than 25 per cent was there any striking subjective improvement following the injection, and objective improvement was either lacking, or minimal and transient in all. The temperature of the inflamed joint was not significantly decreased by the hormone injection. Because cortisone has such a pronounced *suppressive effect on the inflammation of rheumatoid arthritis, it seemed* peculiar that it had so little effect on the joints locally, particularly since topical use of cortisone was so successful in suppressing inflammation in the eye Other workers[4] reported more success with intra-articular injections of cortisone than we had, but even to them the effect seemed too transient to be of practical value in therapy.

Since evidence was accumulating to show that compound F (hydro*cortisone) rather than compound E (cortisone) was probably the* chief anti-inflammatory hormone secreted by the adrenal cortex,[3,9,11,12] we injected hydrocortisone acetate into the inflamed joints of rheumatoid ... made available by Merck and Co. in January

suppression of inflammation was accomplished in 90 per cent of cases within 24 hours after the injection of 25 mg. of *hydrocortisone* into the synovial cavity. The duration of the beneficial effect has varied greatly,

systemic effects from the hormone injection. Successive injections ... a similar effect. After the injections were discontinued, the joint gradually returned to the pretreatment state in most *instances, but in none* was the joint worse than before. Other investigators have confirmed these findings.[13,14,16,20]

... hormone injec-
ed
te
is-
ch
n-

injections was consistent in all these conditions, ...
... nature of the hormone action.

corticotropin is started and continued until reduction of circulating eosinophils indicates a good adrenal stimulation has occurred. Then systematic reduction of the daily dose of corticotropin should be made until it is discontinued. The slow-absorbing, prolonged acting corticotropin is usually very effective in this role

Emotional Readjustment.—The arthritic who has developed a good

better left untreated by these hormones.

OTHER STEROIDS STUDIED FOR ANTI-RHEUMATIC EFFECT

After the remarkable anti-rheumatic effects of cortisone became known it was perfectly natural that other steroids which had chemical composition resembling cortisone should be tested, to see if they possessed significant anti-rheumatic benefits An early concern was the cost—it was hoped that another effective steroid could be made cheaply and in large quantities Later interest centered around the pharmacologic characteristics—the hope being that an inexpensive abundant steroid could be found that possessed potent anti-rheumatic action when administered orally, and little or no other physiologic action, and hence would cause no undesirable effects In search for such an advantageous substance a large number of steroids have been tested Some of these have been referred to earlier Pregnenolone was heralded as an effective "cortisone substitute."[9] However, when carefully studied this interesting steroid was found to possess no consistent effect to augment the action of cortisone or corticotropin, and when used alone it failed to maintain the beneficial effects of these hormones after they were discontinued.[11 14] Many other steroids or "mysteroids"[23] were found to be valueless as substitutes for cortisone. These included· pregnenetriolone, pregnenetriolone diacetate, dihydroxyketo-cyanopregnenedihydrocortisone, 17-hydroxyprogesterone, Reichstein's substance S, 17-ethinylandrostene, 3, 17 diol, androstene, 3, 17 dione, 21-ol, 3, 20-dione allopregnane acetate, testosterone and estradiol[14]

The only steroid other than cortisone which has been shown to have significant anti-rheumatic effect, is 17-hydroxycortisone—hydrocortisone (Kendall's Compound F) This steroid has properties similar to those of cortisone Early reports suggest that hydrocortisone may be required in significantly smaller amount than cortisone acetate to produce equivalent

cortisone, of the many steroids thus far studied, is very striking. That only slight variations in chemical composition so substantially change the

4. In rheumatoid spondylitis, or osteoarthritis of spinal joints. The joints of the spine are anatomically inaccessible to the aspirating needle, so attempts to inject any spinal joints have proved futile

5. Arthritis in joints with no synovial space. Only the diarthroses—the joints with synovial sacs—can be successfully treated by this method. Thus arthritis in the sterno-manubrial joint, in the costochondral junctions or the symphysis pubis is seldom amenable to such local palliation.

6. When injections of hydrocortisone into a joint produce only minimal or transient benefit, on several trials, the method should be abandoned in that case. This is seen in about 15 per cent of patients.

7. In traumatic arthritis with a fracture into the joint, the injection of hydrocortisone may impede the healing of the fracture.

Contraindications to Intra-Articular Hydrocortisone Injection:

1 Presence of infection in or near the joint
2 Multiplicity of joint involvement in rheumatoid arthritis
3 Severe joint destruction, or uncorrected static deformity
4 Arthritis in spinal joints.
5 Arthritis in other than true diarthrodial (synovial) joints.
6 When trial injections have produced little or no benefit.
7 Traumatic arthritis from fracture into the joint

FAILURES AND ADVERSE REACTIONS FROM INTRA-ARTICULAR HYDROCORTISONE

In our experience[1,7] the surprising fact has been that there were so few adverse effects from use of a potent hormone directly into the joint space. Nearly 90 per cent of injections have been followed by definite improvement in the local condition for at least a few days. Many of the poor results were attributable to failure in entering the joint space with the tip of the aspirating needle This was particularly true in injections into the hip for osteoarthritis, where we noted 53 per cent failures. It is imperative that the hydrocortisone suspension enter the joint space, so that it can reach the entire inflamed synovial membrane, or it will have little effect

Following nearly 2 per cent of over 7000 joint injections,[1] there was noted a local *exacerbation* of the joint inflammation. Increased pain, swelling and tenderness came on within a few hours, and persisted up to four days in a few cases Most of these "post-injection flare-ups," however, were mild and lasted only a few hours, following which the condition of the joints improved over the pretreatment state as though there had been no exacerbation.

The incidence of these exacerbations has been unpredictable, since they have been observed after the first, or any one of a series. They have seldom occurred more than once in a given joint, however, so that the occurrence of such a reaction is not a contraindication to further injections. Bacterial cultures taken at such a time have been sterile. For treatment of such flare-ups we have recommended rest, ice packs to the joint, codeine sulfate

many cocktails," *should be regarded* as unfavorable; such changes often forebode major excitement or depression. Insomnia, also, is an important indication of excessive and undesirable stimulation from the hormone, and if continued, the *hormone may lead to mental excitement.* These symptoms should be carefully noted, and their significance appreciated. The [illegible]

[illegible] e done to prevent or reduce tachy- [illegible] nyocardial changes shown in the electrocardiogram, telangiectasis, purpura, thrombosis or hemorrhage, should they occur, except to discontinue the drug.

Should fever or malaise develop in a patient receiving these hormones, very careful study is required to determine whether some infectious process has developed, for the usual symptoms and signs of infection *may be masked.* It is usually wise to discontinue cortisone or corticotropin until the infection has been cured. A serious abdominal disease may progress to perforation of a viscus and development of peritonitis with absence of *usual symptoms of rupture of a viscus or of peritonitis. Slight rise in* temperature or leukocyte count should be regarded with suspicion, and discontinuance of the hormone for a few days may be wise until the true *nature of the situation is apparent and proper treatment instituted.*

Although it occurs infrequently, should development of hypersensitivity to corticotropin occur, it may be of such importance that it has seemed wise to the writer *always* to inject only a small amount (5 mg.) as a test dose whenever starting to use this hormone. If no sign of anaphylactoid reaction occurs within 2 or 3 hours, one need expect no difficulty from subsequent use of this hormone in therapeutic doses This precaution should prevent the occurrence of a severe reaction that might follow the initial administration of a therapeutic dose, it seems particularly indicated before intravenous administration of corticotropin. Skin tests for sensitivity to corticotropin have not been reliable to indicate a hypersensitive state.

POST-TREATMENT PROBLEMS

The "withdrawal syndrome" is the most troublesome of the post-treatment problems. The circumstances which account for this extremely troublesome problem are not entirely understood. Laboratory tests often show that some degree of adrenal-cortical hypofunction exists after discontinuance of cortisone, but *seldom is this hypofunction of major clinical significance,* although it may account for some of the weakness characterizing the withdrawal syndrome.

The many metabolic and hormonal adjustments required after discontinuance of cortisone or corticotropin can be *accomplished more smoothly* if the daily dose is reduced very slowly. Unless complications require quick discontinuance, the patient fares better if several weeks are taken to *withdraw the hormone gradually. After prolonged* treatment, the reduction of the daily dose by 10 to 15 mg. every 10 or 14 days has been found to be a good plan.

6. If deformity is beginning to develop in an actively involved rheumatoid arthritic joint, a *course of hydrocortisone injections* together with proper orthopedic measures and physical therapy can more quickly prevent the deformity, and speed the return of normal function.
7. When *orthopedic correction of deformity* of a certain arthritic joint is being undertaken, whether it be application of traction, wedge casts, or even arthroplasty, repeated local injections of hydrocortisone into that joint can prevent the pain, and reduce the recovery period greatly.

Indications for Intra-Articular Hydrocortisone Injection:

1. When one or only a few peripheral joints are inflamed, providing specific infection has been excluded as the cause.
2. In a few actively inflamed joints even in the presence of more general-
3.
4.
5.
"resistant" joints.
6. To assist in rehabilitation and prevention of joint deformity.
7. As an adjunct with orthopedic procedures.

CONTRAINDICATIONS TO USE OF INTRA-ARTICULAR HYDROCORTISONE

It cannot be stressed too often that *intra-articular hydrocortisone injection is a palliative, local, and temporary treatment,* even though it can be repeated apparently indefinitely. As a result of trial and error clinical use for nearly two years, we conclude that the following conditions either contraindicate the use of this method, or at least render its use impractical:

1. The prime contraindication to local use of hydrocortisone is the presence of infection in or about the joint. Tuberculous arthritis of an ankle, though temporarily improved after injection, became much more severe after such treatment in our experience. It is reasonable to avoid injection of a hormone, known to reduce reaction and resistance to the spread of infection, into the site of such an infection. It may be possible after considerably more research, to combine the local injection of hydro- [illegible] treating infected joints, but for the

[illegible] olves *many* joints actively, the injection of hydrocortisone into one or two is usually futile.

3. When severe or late osteoarthritis is secondary to a static deformity, or has itself produced severe deformity, *the injection of hydrocortisone* can accomplish little, unless preceded by adequate corrective measures. Likewise in joints markedly deformed by rheumatoid arthritis, intra-articular hydrocortisone can help but little.

manner of action of these potent hormones has not yet been elucidated The effects are temporary, suppressive but not curative, and there is no evidence that the ultimate course of a *chronic* rheumatic disease is favorably altered by these hormones, as they have been employed. There are potential dangers involved in using these materials, great care and definite precautions must be taken These hormones can be put to practical use in treatment of patients, but the significant potential complications and limitations in their usefulness have thus far kept these agents from being *routinely* used to treat inflammatory rheumatic diseases. Indeed, the use of these hormones has become quite highly selective and for the purpose

Troubles must be expected to occur in some patients, and the physician should be prepared to meet them One should consider not only what may happen *during* the use of these hormones, but also what might occur *after* their use

this new addition to important scientific knowledge has provided, research will continue to increase manyfold and will be undertaken with greater [illegible]igor and new enlightenment. It would be unjust not to expect newer [illegible]nvestigations to solve the problems which remain, so as to provide the [illegible]neans of prevention or more successful treatment of these prevalent and [illegible]erious rheumatic diseases Never before has the hope for success been [illegible] justified and the future of rheumatology been so bright!

BIBLIOGRAPHY

[illegible] 147, 1538, 1951

[illegible] J Am. Med Assoc, 14[illegible], 1951

Conn, J W., Louis, L. H, and Fajans, S S Science, 113, 713, 1951

Dobriner, K.: Address, A[illegible] Meeting American Rheumatism Association, Atlantic Ci[illegible], June, [illegible]

Freeman, [illegible] Am [illegible] Assoc, 142, 1124, 1950

Freyberg, [illegible] et al Science, 112, 429, 1950

[illegible]n Dis, 10, 1, 1951.

[illegible] 1538, 1951

[illegible] 412, 1951

[illegible]nson, C, and [illegible]son, M Symposium [illegible]linical Practice [illegible], Blakiston Co, Phila,

[illegible]stein, R. [illegible] New England Med

[illegible]ally, E Gastroenterology, 16,

Dustan [illegible] Am Med Assoc, 146,

when the symptoms have returned. Should the beneficial effect continue for three weeks, reinjection is usually withheld until needed For extremely active joints, however, some workers[16] have given several injections on successive days to increase the original effect. We have given as many as 30 injections into a single joint, repeating only as often as symptoms recurred. In most patients the succeeding injections have duplicated the degree and duration of relief afforded by the first injection, but in some

average duration of relief from each injection is about 8 days, in osteoarthritis, about 2 to 3 weeks, in traumatic arthritis and self-limited conditions subsequent injections may not be necessary. The average dose for a single joint is 25 mg of hydrocortisone acetate suspension, less in smaller joints, and more in larger joints The dose for individual joints is given below in the description of technic for injection of each If the suggested dose does not seem adequate, a larger dose may be tried the next time, but we have found that the response is not necessarily in proportion with the amount used, and larger doses than needed may actually decrease the benefit.

THE TECHNIC FOR INTRA-ARTICULAR INJECTION

Any arthritic peripheral joint may be injected, if indicated, and also the temporomandibular, acromioclavicular, or sternoclavicular joints. The anatomic structure and depth below the body surface of the spinal joints makes local attempts to treat such articulations impractical

An anatomical atlas, and an x-ray film of the joint being aspirated should

into the general circulation.)

The technic for joint paracentesis and injection is fairly simple and clear-cut. The *object* is to puncture the synovial sac, aspirate excess fluid, and inject the hormone with as little pain as possible, with minimal trauma to the joint and adjacent structures, and without introducing infection into the joint cavity

The optimum site for paracentesis is, usually, the location on the extensor surface where the synovial pouch is closest to the skin, and remote as possible from major nerves, arteries or veins. The usual site for needling the specific joints most often treated will be given in detail below.

Materials Needed.—A small office tray containing an antiseptic solution, alcohol sponges, 1 per cent procaine solution, hydrocortisone acetate suspension, two 24 gauge sterile needles 1½ inches long, two 20 gauge sterile

biologic action of the steroids is very interesting. Inspection of the structure of various steroids (Figure 91) indicates that for anti-rheumatic activity, several details are required; absence of any one alone will result in the *absence of significant anti-rheumatic action.* These requirements are:

1. OH at carbon 17.
2. Double bond between carbons 4 and 5.
3. Double-bonded O at carbon 3.
4. Double-bonded O, or OH, at carbon 11.
5. OH, or its ester, at carbon 21.

Cortisone and hydrocortisone are the only steroids known which fill all of these requirements.[26]

however, such effect has not been consistent, and since insulin must be injected, this would be impractical for general application even if it were helpful. Para-aminobenzoic acid orally, in large amounts together with

No satisfactory substitute for cortisone and hydrocortisone has been discovered, nor have practical, dependable means of augmenting their benefits and decreasing the non-antirheumatic effect been found.

LOCAL USE OF HYDROCORTISONE

In the spring of 1950, Thorn[45] *and* Hollander[23] reported separately that cortisone acetate was sometimes effective to reduce symptoms and signs of inflammation when injected directly into the joint. The writer and his collaborators found quite consistently that temporary benefit occurred when 25 to 50 mg of cortisone acetate were injected intra-articularly into

Hollander *et al.*[24] reported much better results from the intra-articular

r, have had

h more con-

sistent benefit, without producing significant local irritation, than has

ion of

tisone

(*See*

Chapter 18)

SUMMARY

Cortisone, hydrocortisone, and corticotropin have potentialities for producing remarkable benefit in various types of rheumatic disorders The

½ grain by mouth, and, when effusion is marked, aspiration of the joint for relief of tension.

Following less than 1 per cent of hydrocortisone injections into arthritic joints in our experience, there occurred a peculiar *weakness* of the extremity treated. The patient described this as a "heaviness" of the leg or arm, which persisted from one to three days. There was no instance in which this weakness persisted longer, and seldom did this occur more than once. In addition to this local feeling of weakness, 0.3 per cent of the injections were followed by a spell of generalized weakness, malaise, and vertigo which persisted for one to three days. Neither of these phenomena have as yet been satisfactorily explained, but since their occurrence has been *rare, transient, and seldom recurring,* they have not been particularly troublesome.

One patient developed local urticaria following injection, which was found to be due to allergy to the aqueous vehicle of the hormone suspension. Several other patients were found sensitive to procaine, and developed a local wheal following its use for local anesthesia.

Although we have made it a point to give the intra-articular hydrocortisone injections as an office or clinic procedure, using neither drapes, rubber gloves, or any other than routine sterile precautions, only one joint infection was produced by the procedure in over 7000 injections. A staphylococcic infection was produced in a knee joint. This was detected 24 hours after injection, in a patient who was taking 100 mg. of cortisone by mouth daily for rheumatoid arthritis. The inflammation in the knee joint had not been adequately controlled even by this large dose of hormone. The injection had been given inadvertently through an area of the skin on which were *several pustules of acne. Although a skin antiseptic* was used, the infection was carried through to the joint by the aspirating needle. A single injection of 100,000 units of penicillin into the joint checked the infection, and subsequent hydrocortisone injections have been given without adverse effect.

Since there is a potential danger of producing bacterial contamination of the joint space by this procedure, particularly in patients receiving cortisone or ACTH systemically, special care should be taken in such instances. Holbrook[5] has suggested that an intramuscular injection of 300,000 units of penicillin be given all such patients each time intra-articular injections are made. This precaution is certainly merited, since the resistance to infection is lowered in cases receiving systemic cortisone or ACTH therapy. Penicillin should also be given at any time that the physician feels his sterile technic might not have been perfect, or that a post-injection flare-up might be an infection of the synovial sac.

If the physician is careful to preserve surgical cleanliness in his technic, he need have little fear of infection of the joint, since this has occurred *only once in 7000 injections* in our experience. As mentioned previously, however, hydrocortisone should *never* be injected into a joint already the site of infection.

ADMINISTRATION AND DOSAGE

The technic for injection will be described in a subsequent section. The plan of treatment should be arranged so that no more than two or three

INJECTION OF SPECIFIC JOINTS AND SUGGESTED DOSAGE

Since the technic for paracentesis of individual joints has received little attention, and includes many specific problems, a description for each of the joints frequently injected is given here.



A



B

FIG 97.—*A*, Injection into the knee *B*, X-ray showing needle in place under medial side of patella.

CHAPTER 19

PREVENTION OF DEFORMITIES FROM ARTHRITIS

By John G. Kuhns, M.D., and Theodore A. Potter, M.D.

The multiplicity of symptoms of rheumatoid arthritis makes it difficult for one physician to comprehend the problem intelligently and to treat it effectively. The inflammation in the joints with the subsequent deformities

of the internist and orthopedic surgeon.

The orthopedic management of rheumatoid arthritis includes many procedures. One of these is the supervision of the patient in positions of lying, sitting and standing. Stiffness and deformity can arise from gravity and from prolonged assumption of positions which favor deformities. A second factor in the orthopedic management of chronic arthritis is the maintenance or teaching of good posture. Faulty posture is found in almost every patient suffering from chronic arthritis. Its correction aids in preventing recurrence of disability in arthritis. A third factor in orthopedic treatment is the maintenance of strength and efficiency in the muscles and ligaments and the prevention of deformities.

A fourth part of orthopedic aid in chronic arthritis is the correction of deformities and disabilities by apparatus, manipulation and surgical intervention. These procedures are used only after the acute stage of the arthritis has passed. The fifth and final stage is assistance in all that is comprised in the rehabilitation of the patient. Procedures for the correction of deformities will be described in the next chapter, and rehabilitation in Chapter 23.

Positions.—Usually the patient with acute arthritis is confined to bed

knees while sitting to relax the hamstring tendons. For reading, an inclined plane of pillows can be made by placing one large pillow crosswise at the head of the bed, another large pillow is placed over this lengthwise and a

needles $1\frac{1}{2}$ to 2 inches long, a sterile 5 cc. syringe for injecting procaine, a sterile 20 cc. syringe for aspirating joint fluid, one or two sterile 2 cc. syringes for injecting hydrocortisone, a few gauze pads, and prepared small patch dressings (*e. g.*, Band-Aids) constitute the total equipment needed Culture tubes and a graduated glass cylinder for measuring synovial fluid may also be included. No sterile drapes or rubber gloves are necessary

Method.—After the optimum site for insertion of the aspirating needle has been chosen, the skin is cleansed twice with alcohol, or with antiseptic solution and then with alcohol. *If there is marked effusion present it is* seldom necessary to infiltrate the tissues with procaine, as the introduction of the needle will be quick and easy. In some individuals a brief spraying [illegible] …lution [illegible] he needle [illegible] synovial membrane. *The tip of the needle is freely* movable when the joint space is entered—it may even grate over the cartilage without pain to the patient. If excess fluid is present, it may then be aspirated into the syringe.

Entrance into a distended synovial sac is as easy as puncturing an inflated balloon, but entrance into a sac not distended may be as difficult as puncturing a deflated balloon—the relaxed synovial lining may push ahead of the needle point. Folds of synovium or cellular debris may act as a flap-valve on the end of the needle, preventing easy withdrawal of fluid. When this is suspected, the needle should be moved about gently, and a little of the fluid already withdrawn reinjected to push the fold or clot away from the tip of the needle.

Aspiration may be facilitated by wrapping the joint, except for the site of paracentesis, with an elastic bandage to compress the free fluid into that portion of the sac being punctured. It is well to aspirate all readily accessible fluid at the time of paracentesis.[16]

[illegible] is then detached, allowing the needle to remain [illegible] to the aspirating needle, still in situ *If more than gentle pressure on the plunger is required to inject the solution, the needle is probably not free in the joint space, and should be readjusted.* Even if no fluid has been obtained, the easy movability of the needle tip, and the easy introduction of the hormone suspension can give assurance that the tip is in the joint cavity If fluid is present, the injected suspension may be mixed with the synovial fluid by a barbotage-like repeated withdrawal and reinjection, or "pumping."

The needle is then withdrawn, the puncture site is wiped with an alcohol sponge, and covered with a small sterile dressing. If much fluid has been aspirated from a weight-bearing joint it is often advisable to apply an elastic bandage, to reduce the instability resulting from the now relaxed joint capsule. The patient may resume normal activity immediately, but he should be cautioned not to abuse the joint, even though pain and swelling disappear.

for aspirating specific joints In addition, there has grown in the minds of most physicians a fear of aspirating joints because of the possibility of thereby introducing infection. Based on the experience of performing thousands of joint aspirations for diagnosis, in research, and for hydrocortisone injection into joints, we can state with confidence that there is little danger, providing ordinary aseptic precautions, such as observed with any hypodermic injection given in a clinic or physician's office, are observed. Joint paracentesis and injection is as simple and safe a procedure as

as

in

injecting a certain joint.

As skill in venipuncture is acquired by practice, it is necessary to attempt joint paracentesis often enough so that it becomes simple, quick, and painless to the patient.

While it now appears established that intra-articular injections of hydrocortisone offer fairly

for many types of

servations over a m

the ultimate effect of this application of hydrocortisone. *Only time will tell whether the alleviating effect of this therapy will result in the long-range benefit the physician seeks for his patient.* An observation period of only 2 years is insufficient for such evaluation.

BIBLIOGRAPHY

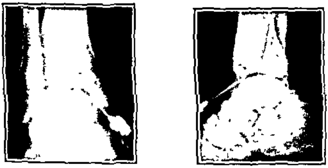
1. Brown, E M, Frain, J B, Udell, L., and Hollander, J. L: Am. Jour Med (In Press)
2. Colonna, P C: Regional Orthopedic Surgery, Philadelphia, W. B Saunders Co, 1950
3. Science, 113, 713, 1951.
4. H, Durivage, J, and Traeger,
5.
6. Hollander, J L Bull Rheum Dis, 2, 3, 1951.
7. Hollander, J. L, Brown, E. M, Jessar, R A., and Brown, C Y.: Jour Am Med. Assn, 147, 1629, 1951.
8. Hollander, J L, Zacco, M, and Dohan, C. H.. In preparation.
9. Mason, H L Proc. First Clin. ACTH Conf, Ed by Mote, J. R, Philadelphia, The Blakiston Co, 1950
10. Mawson, R Brit Med. Jour., 2, 691, 1946
11. Proc Second Clin ACTH Conf,
12. ACTH Conf,
go, June 1952
13. o, June 1952.
14. ery, St. Louis,
15.
16. eum Dis, 11, 112, 1952.
17. Communication
18.
19. of Am Rheum. Assn, Chicago,
20. 2.

adequate dose

Hip.—This is the most difficult joint to aspirate and inject, mainly because of the amount of soft tissue overlying it in all directions In many cases of osteoarthritis of the hip it has been impossible to enter the joint space with certainty, even under fluoroscopic guidance, because of the flattening of the femoral head and osteophyte formation about the ace-



A



B

C

FIG 98 —*A*, Injection into left ankle—antero-medial approach *B*, X-ray, showing A-P view of needle in place in right ankle *C*, X-ray, showing lateral view of needle in right ankle

flected in more efficient functioning of the body, decreased inflammation in the joints, and indirectly in better health.

The usual effects of faulty posture can be grouped as follows: increased fatigue, sprain, traumatic arthritis, deformity and visceral disturbances.

[illegible]

traumatic arthritis. Changes can take place in the bones and ligaments of these individuals whose bodies are badly aligned and lasting deformities may develop.

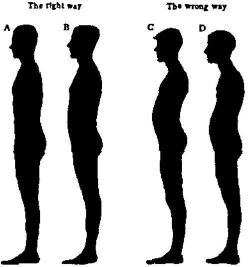


Fig 105 —Chart of good and bad posture (Harvard Chart of Body Mechanics or Posture)

The visceral disturbances which may accompany poor posture vary greatly in different individuals They usually are more serious in those of slender [illegible]
These [illegible]
Throug[illegible]
assimilation and excretion may be slowed down and made less efficient

These effects which would be scarcely noticed by an active apparently [illegible]

[illegible] visceral disturbances are extremely common in chronic arthritis and any means that may improve their function is a welcome aid in treatment

Knee.—The largest synovial cavity in the body, and a weight-bearing structure, the knee is also one of the commonest sites of synovial inflammation. It is the easiest *joint to aspirate and inject* A *tightly distended* knee joint can be aspirated from almost any angle without difficulty, but the following technic may be used for ready entrance into the joint space even if there is little or no excess fluid present.

The patient lies on a table with the knee fully extended. The site for puncture is on the antero-medial surface, 1 to 2 cm. medial to the inner border of the patella. The needle is inserted in a lateral and slightly posterior direction in a line aiming to slide between the posterior surface of the patella and the patellar groove of the femur. In this way the needle tip may crepitate on the undersurface of the patella, demonstrating entrance into the joint space even though no fluid can be withdrawn. Figure 97 shows the position of the needle. The advantages of this approach are that there are seldom folds of synovial membrane or pannus

If the patella is ankylosed, or there is marked flexion deformity of the knee, aspiration may be accomplished by puncture antero-posteriorly beside the inferior patellar tendon, through the fat pad into the knee joint space between the condyles of the femur. Puncture of the knee through the popliteal space is not advisable, because of the large popliteal vessels A Baker's cyst, or posterior herniation of the knee joint capsule, may be aspirated directly, however, if it is superficial and distended. Twenty-five to 37.5 mg. of hydrocortisone suspension are the usual dose for injection into a knee.

Ankle.—As shown in the illustration (Fig. 98), an antero-medial approach *is usual* *Procaine infiltration is often advisable prior to ankle aspiration* The needle is aimed at the tibio-astragalar articulation from a point 1 cm. above, and 1 cm lateral to the internal malleolus, just medial to the extensor pollicis tendon. The needle tip must penetrate about 3 cm. inward and ... of the ankle joint capsule, and then

on the plunger of the syringe ...
probably in ligament or cartilage. Twenty-five mg. is the usual dose of hydrocortisone used for ankle injection.

Tarsal Joints, and Tarso-metatarsal Joints.—A roentgenogram of the ... in the location best suited for entrance ... used, and procaine anes- ... perficial location of the ... election, rather than the plantar surface. The needle may have to be "teased" down between the cartilages, and fluid can only rarely be aspirated Here the freely movable needle tip, the depth of penetration, and the lack of force needed to inject the hormone suspension, assure that the joint space has been entered *Fluoroscopic guidance is very helpful when small joints are injected.* Ten to 15 mg. of hydrocortisone is the usual dose for such joints.

Toes.—After procaine infiltration, a 22 gauge needle is inserted from the dorsal surface, either medially or laterally, sliding beneath the extensor

with the hips firmly against the back of the chair. In wheel chairs or soft chairs a pillow can be placed behind the low back; this will hinder slumping. *The patient should not sit in easy chairs or wheel chairs for long periods of time*

Good Posture.—Good positions in convalescence are part and parcel of habitual good use of the entire body. *Body mechanics has been defined as* the mechanical correlation of the various systems of the body with particular reference to the skeletal, neuro-muscular and visceral systems It is a dynamic concept of posture, of the body in efficient use. It means good alignment of the entire body while the body is being used. *Faulty posture* favors the development of deformities and leads to spasm and disalignment of joint surfaces. This latter produces further inflammation in joints that are already inflamed.

The causes of poor posture are many. In man's development from the plantigrade to the orthograde type of progression, he did not inherit an efficient mechanism for balance. Balance comes only as a series of conditioned reflexes. In infancy, malnutrition, chronic illness, tight clothing and faulty furniture are important factors in bringing on poor posture. Later carelessness, sedentary occupations, obesity and the degenerative processes incident to increasing age help to make the body posture worse. Poor posture, which is an almost universal finding in patients, becomes worse with the development of arthritis. A vicious cycle is set up; the individual becomes more fatigued and slumps more and this in turn increases the fatigue.[31]

There are various ways of determining whether the body posture is good or not. Common signs of poor posture are a head held far forward, round shoulder and flat chest, prominent abdomen and marked lumbar lordosis. [illegible]

recumbent patients and to those who are seriously disabled. For these a different method of judging posture must be used. The following are the usual criteria of habitual good posture

[illegible] in the anteroposterior curves of the spine

[illegible]

costal angle should be a right angle.

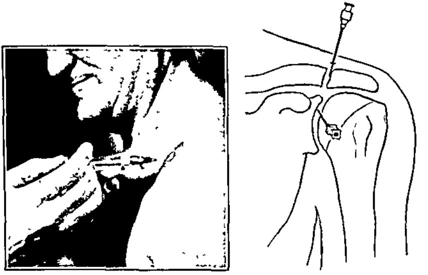
3. The greatest circumference of the body should be at the xyphoid in all positions of the body.

4. The lower abdomen should be flat and the upper abdomen should be full and rounded. In this position the diaphragm can work effectively and there is no pressure upon or crowding of the abdominal viscera.

These criteria are the goal for which one works during the treatment of chronic arthritis Often fixed deformity prevents the physician from attaining good posture completely, but whatever improvement is gained is re-

... The tip is then slightly withdrawn
... case injection meets little
... sual dose of hydrocortisone
for the hip is 37.5 mg.

The lateral approach to the hip joint is also difficult, but has the advantage that the needle "follows the bone" to the hip joint. A lumbar puncture needle is inserted just anterior to the greater trochanter, in a sagittal direction, pointed toward the middle of Poupart's ligament. The needle tip slides in anterior to the periosteum of the neck of the femur, and enters the joint space anteriorly and near the upper reflection of the synovial sac. (*See* Fig. 99*B*.)



A *B*

FIG 100.—*A*, Injection of the right shoulder joint. *B*, Diagram showing position of needle for injection of shoulder joint and for acromio-clavicular joint.

Shoulder.—Aspiration of the shoulder is easily accomplished by an-

...

nearly always bulges anteriorly. Twenty-five mg. of hydrocortisone is the usual dose for shoulder injection.

Acromio-clavicular Joint.—The plane of the joint can most easily be

...

directly anterior to the joint. Because of the fibrocartilaginous articular disc, it is a rather difficult joint to inject. Fifteen mg. of hydrocortisone is the usual injection dose.

of the head or on the iliac crest. Eventually standing, foot and walking

head erect with chin held in.



Fig 106 —*Hyperextension* to expand the chest, raise the diaphragm, increase abdominal breathing and circulation and correct round shoulders Use this position for one-half hour after meals, a pillow under dorsal spine and a pillow under the knees (to relax the muscles of the back); pillows must not be kept under the knee for long periods or contracture deformities may result (Swaim, courtesy of Am Jour Nursing)



Fig 107 —*Face prone position*, support under the abdomen to flatten the lower back (Swaim, courtesy of Am. Jour Nursing)



Fig 108.—Postural exercises performed in recumbency The hands are under the head, the abdomen is flat, the knees are flexed

tabulum. Occasionally, even in osteoarthritis, fluid may be aspirated

cm. lateral to the femoral pulse, depending on the size of the patient. (*See* Fig. 99.) The needle is inserted at an angle of 60° with the skin, pointing postero-medially, through the capsular ligaments, which are

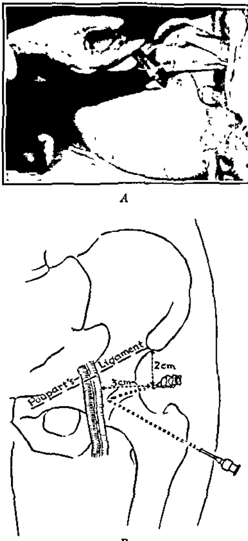


FIG 99.—*A*, Injection of left hip. Crescent shows location of anterior superior spine of ilium; line is over Poupart's ligament *B*, Diagram shows location of structures about left hip, and two directions of approach for injection

Improvement in posture is attempted throughout the treatment of the chronic arthritis. In the early stages the patient may be recumbent all of

tient and are modified when fixed deformities are present. Usually the first two or three exercises are prescribed several times twice daily and are performed more times and others are added as the patient's ability to perform them increases.

Positions and Exercises for Improvement of Posture of the Bed Patient
Keep the patient on a firm bed A fracture board should be placed under the mattress. Permit the use of only one small pillow under the head.
e back, no pillow pillow under the this position for twenty to thirty minutes after each meal. Lessens drag upon abdominal viscera, elevates ribs and diaphragm, flattens lumbar spine and tilts pelvis backward. The position of hyperextension will promote better circulation in the abdomen and extremities.
extension position.
lower abdomen in
Lying on the back, knees flexed, hands behind the head: pull in the
buttock muscles
ly separating the ribs from the pelvic bone Stretch the opposite side.
Same position—Place the hands on the lower rib margins anteriorly; breathe deeply and pull the lower rib margin up and out; maintain this pull while exhaling.
These exercises are performed 3 to 10 times twice a day, depending upon the strength of the patient.

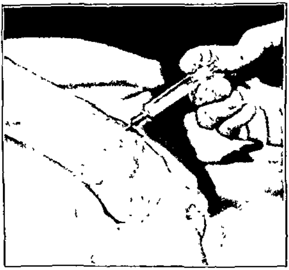
At the same time an effort is made to correct postural deformities by special positions, as described in the list above. By these positions postural deformities can be corrected gradually.

As the patients improve in well-being and acquire ability in performing the exercises lying, sitting exercises are given The patient sits on a firm

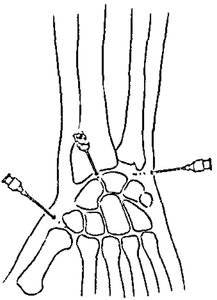
r ab-

back

medial direction at a point just distal to the lateral epicondyle (capitulum of the humerus), and just proximal to the head of the radius, to a depth of about 2 cm, when the point of the needle feels free in the joint. Aspiration of fluid from the elbow is more difficult with this latter approach.



A



B

. —*A*, Injection into the wrist *B*, Diagram showing location of needle for roach, ulnar approach, and for injection into carpometacarpal joint of left

motion five to ten times several times a day. As strength increases more strenuous exercises are given. To regain normal strength after inflammation has gone from the joint, resistive exercises (weight lifting) are given. The uninvolved joints should not be neglected, and should be carried through a full range of motion at least several times daily. (*See also* Chapters 21, 22, 23.)

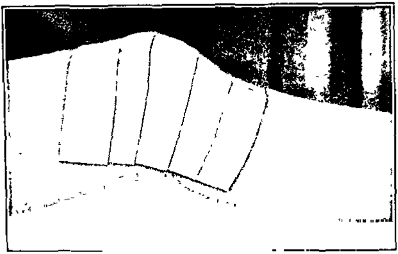


Fig 111 —Adhesive strapping of inflamed knee

SPLINTING OF AFFECTED JOINTS

When joints are swollen and painful on motion they should be protected.[2] The amount of protection is dependent upon the severity of the inflammation. The amount of muscular spasm determines the rigidity of the support necessary. If there is only a slight amount of swelling and little pain on motion a firm bandage about the point may be adequate splinting.[8,15] The most comfortable type of bandage is an elastic cotton bandage (ACE bandage). Felt strips or sheet cotton rollers can be applied under the elastic cotton bandage to add to its security. On small joints a bandage 2 inches wide is most effective. Wider bandages are used for the larger joints. Such bandages are effective as a temporary support during convalescence, when full recovery is expected in a few weeks. It is also used when the muscles are not quite strong enough about the joint. Bandages are not

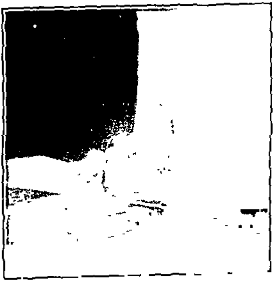
... plaster
... sprains

which come in joints when there is articular damage or deformity. This is common in arthritis. The joints most commonly protected are the wrist,

Elbow.—The elbow may either be held at 90°, or fully extended, the latter position in order to distend the capsule as tightly as possible. The bulge of synovium, when effusion is present, is noted on the extensor surface, lateral to the olecranon process of the ulna. The aspirating needle should be inserted distally just lateral to the olecranon and just below the lateral epicondyle of the humerus. (Fig. 101.) Often, particularly in injecting the elbow for "tennis elbow," the lateral approach directly into the radio-humeral articulation is desirable. The needle is inserted in a



A



B

FIG 101.—*A*, Injection of right elbow in position of maximum extension *B*, X-ray of left elbow showing needle in place with elbow at 90°. Needle is inserted *lateral to* olecranon process of the ulna.

flatten the lumbar spine and to push the lower abdomen in and up In

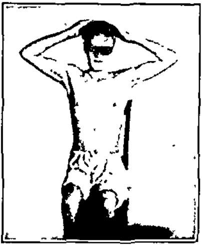


Fig 109.—Postural exercises performed while sitting The back is flat, the abdomen retracted The hands are on the head or resting on the [illegible]

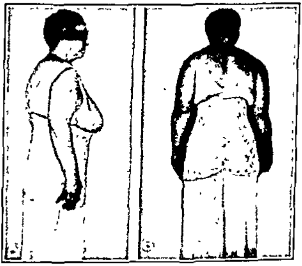


Fig 110 —Corset used to aid in maintaining good posture The abdomen is pulled in the low back is flat. *A.* Lateral view. *B.* Posterior view.

women this can sometimes be done by a properly fitting corset. Such apparatus is worn while the patient is up and is *removed for exercises* and at night. *It is no longer worn when good body posture can be kept without external support.*

1.3 cm. [illegible]
slightly [illegible]
1.5 cm. [illegible]
cartilagi[illegible]
sure the [illegible]
the sup[illegible]
proved an adequate dose.

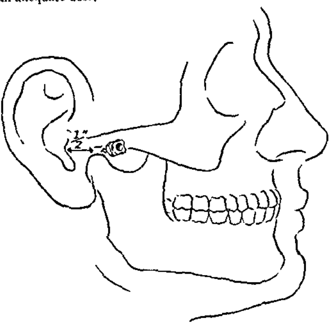


FIG 104 —Diagram showing location of needle for injection of the temporomandibular joint.

Usual Effective Dose of Hydrocortisone Acetate per Joint Injection:

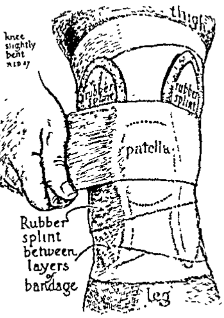
Joint	*Dose*	*Joint*	*Dose*
Knee	25–37 5 mg	Shoulder	25 mg.
Ankle	20–25 mg	Elbow	20–25 mg
Tarsal	10–15 mg	Wrist	15–25 mg
Metatarso-phal	10 mg	Metacarpo-phal	5–10 mg
Interphal toes	5 mg	Interphal fingers	5–10 mg.
Hip	37 5 mg	Acromio-clavicular	15 mg.
Temporomandibular	10–15 mg	Sterno-clavicular	15 mg

CONCLUSION

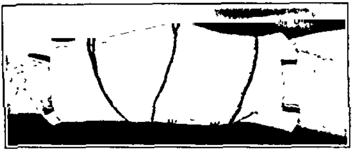
The technic of joint paracentesis, probably because of its simplicity, is scarcely more than mentioned in most textbooks Even textbooks on operative orthopedic surgery[7,11] do not sufficiently describe the methods

... these latter have never come

motion in the joint be prevented, muscular spasm quieted and comfort assured to the patient.



A



B

FIG 112 — A, Combination *sponge rubber and elastic bandage*, partly applied to the left knee, featuring the shape and position of the rubber sponge splints To complete the support, the bandage is continued to the top edge of its inner layer (Truslow, courtesy of Jour Am. Med Assn) *B*, Instead of sponge rubber, strips of thick felt 1¼ inches by 8 inches can be applied to the sides of the knee joint beneath the ACE bandage

Paracentesis medial to the olecranon [illegible]
ti [illegible]
[illegible]
([illegible]
point just distal to the radius, and just ulnar to the "anatomic snuff box" If the needle can be easily inserted to a depth of 1 to 2 cm. and feels free, it is probably in correct position. Most of the small *inter-carpal joints* have connecting synovial spaces If there is marked synovial bulging on the ulnar surface of the wrist, the joint may be entered by the insertion of the needle just distal to the ulnar styloid process, and dorsal to the pisiform bone (Fig. 102 B). Fifteen to 25 mg. of hydrocortisone is usually an effective dose.



FIG. 103 —Injection into a proximal interphalangeal joint of finger.

Carpo-metacarpal Joint of the Thumb.—This small joint is frequently troublesome in osteoarthritis, and does not communicate with the other joint spaces of the wrist. It may be entered dorsally from the radial side of the hand, as shown in Figure 102 B. Five to 10 mg. of hydrocortisone is usually an adequate dose.

Metacarpophalangeal and Interphalangeal Joints.—These are easily punctured from the lateral or medial approach on the *dorsal surface.* A small needle is desirable for such injection (24 gauge) to avoid undue trauma, unless aspiration of fluid is necessary. It is frequently necessary to "tease" the needle between the cartilages, at the same time making traction on the finger to pull the joint cartilages apart. Five to 10 mg. of hydrocortisone is adequate for injection into such small joints.

Temporomandibular Joint.—It is sometimes desirable to inject hydrocortisone into this joint for rheumatoid arthritic involvement, for osteoarthritis, or for "clicking jaw." A roentgenogram of the area is necessary for reference. The needle to be used for injection should be small (23 or 24 gauge). It is inserted at a point just below the zygomatic arch, about

elbow, low back, knee and ankle. Strapping never entirely encircles the joint, going about two-thirds around it. It always covers and com-

fore applying the adhesive plaster and a gauze bandage is put on over the adhesive strapping for twelve to twenty-four hours to prevent slipping of the adhesive. The adhesive strapping should not be immersed for long periods in water or it will loosen. Sponge baths or shower baths usually do

often prevent further injury to the joints. They do not prevent or correct deformities

A useful type of strapping, particularly about the knee, ankle, and elbow combines elastic adhesive dressing with pressure. This gives more support than the two previous types of bandaging Either an elastic form of adhesive tape, or zinc gelatin paste on gauze may be used

A somewhat more effective form of splinting of inflamed joints is *traction* [21] This can be used both in the upper and lower extremity to relieve muscular spasm and correct mild flexion deformities In arthritis it is used most commonly on the hip and knee joints. In the upper extremity it can be used on the elbow and finger joints, rarely at the shoulder On the lower extremity 2-inch wide strips of adhesive, preferably moleskin adhesive plaster, are applied to the medial and lateral sides of the leg over skin shaved and painted with tincture of benzoin. The bony prominences about the knee and ankle are padded with felt or sheet cotton The foot

traction rope. A splint or pillow is sometimes applied at first to secure traction in line with the deformity. To be effective traction must be continuous Intermittent traction gives only temporary relief of pain. A useful temporary form of traction is to apply coarsely woven cloth or strips of sponge rubber to each side of the leg and wind an elastic cotton bandage firmly about it. This will take 5 to 10 pounds of traction and can be removed from the leg quickly for other treatment simply by unwinding the bandage.

If greater traction is desired it can be secured in the following ways A Kirschner wire can be inserted through the femoral condyles, upper tibia or os calcis and traction up to 20 pounds can be applied through a stirrup attached to the Kirschner wire. The atrophied bone in chronic arthritis will rarely stand more than 20 pounds of traction. Traction is not comfortable, must be supervised constantly and on the whole is much less effective

When soaked in water, plaster of Paris takes up water and reverts to the solid crystalline state with a rapid formation and interlocking of crystals.[20] [illegible]

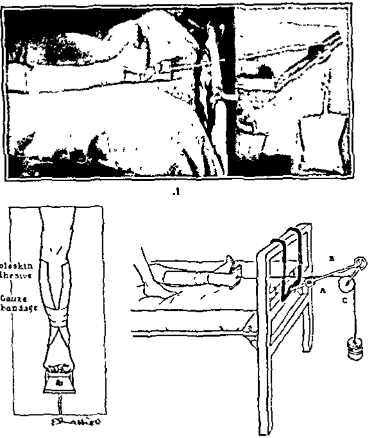


FIG 114 — A Removable knee-traction traction. Sponge rubber attached to traction straps is firmly bound on each side of the lower leg with a bandage B Usual form of adhesive traction for the knee

[illegible]

wrap fresh plaster over a dry plaster surface, one should *first roughen* the dry surface by scratching this with a sharp knife. In this way also a broken plaster may be patched with strips of freshly wetted plaster.

Good plaster technic includes use of a minimum of plaster, rapid application of the cast and protection and comfort to the patient One usually

Prevention of Flexion Deformity of the Knees

Never place pillows under the knees for comfort in these patients; this is the commonest cause of such a flexion deformity.[11]

Use a posterior molded splint or cast extending from the upper part of the thigh to the toes with the knee in full extension A full cast including the foot is best

Incorporate a wooden strip into the leg splint posteriorly at the ankle to control rotation of the hip joint and prevent outward rotation of the lower leg

Remove the splint once daily and allow active movements

Apply hot fomentations or other heat to the knee twenty to thirty minutes several times a day

Employ muscle setting and non-weight-bearing exercises until the acute inflammation has subsided *

With improvement, lessen the period of splinting gradually so that within a short time, splinting may be required only at night, until inflammation has subsided

Even in the later stages of the process, permit the splints to remain in place all night as flexion of the knees is common during sleep (unless prevented by splints)

Do not permit weight bearing until the acute inflammation has subsided entirely and a period of exercises without weight bearing has produced no harmful effects, the leg muscles must be in a condition to support this weight. **The patient should not walk on a flexed knee joint,** *or he will produce distortion in both lower extremities and in the trunk* He should not be allowed to walk [illegible]

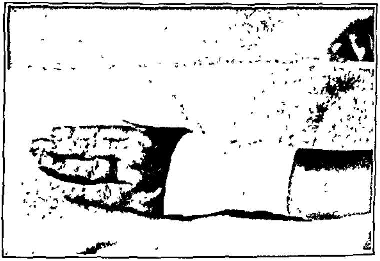
[illegible]

One of the fears of the patient with acute joint involvement is excruciating pain which may occur on slight motion of the joint With the joint held firmly in a bivalved cast painful movement of the joint does not occur. If all movement is painful, muscle-setting exercises can be taught These can be carried out without motion in the joint. No passive motion should be done. Following this the splints are reapplied. As long as joint pain, tenderness and swelling persist, it is advisable that splints be used for the affected joint during most of the day and night so that muscular spasm will not cause deformities [24]

The commonest deformities of the knee are flexion, subluxation and outward rotation. The cause of this flexion deformity is the desire of the patient to relieve the pain in the acute stage of the disease. Flexion of the

Usually the plaster need only be kept on the limb for a few days as a solid encasement. After a few days the plaster casts for the lower extremities are cut into anterior and posterior halves. This permits removal of the anterior half and later of the entire cast for short periods of the day, for heat, exercises or other treatment.[19] A single plaster on the volar surface fastened with a bandage is usually all that is required in the upper extremity.

Plaster of Paris Splints.—Molded plaster of Paris splints are best [illegible] These should be light but thick enough to prevent breaking of the splint [illegible]
The plaster is [illegible]
placed on the [illegible]
home, strips of impregnated plaster will prove useful These are sold already cut in various lengths, which need merely to be immersed in water, quickly withdrawn and applied to the part involved. These are available



[illegible] applied

in several different sizes—3 x 15, 4 x 15 and 5 x 30 inches, in boxes of 50 sheets each. From 7 to 10 layers (or sheets) may be needed for each splint depending upon the joint involved. It requires not more than a few seconds for the splints to become saturated in water; the plaster sets within eight to ten minutes.

When Applying Plaster:

1. Pad all sharp, bony prominences which may be covered malleoli, anterior-superior spines, crest of the ilium, knee, etc If the patient is thin and the spinous processes are prominent, use padding over these
2. If all prominences are padded, additional padding elsewhere is unnecessary.
3. If the ribs flare, these can be padded.
4. Avoid pressure from fingers.

knee is the most comfortable position when swelling is present as it lessens the pull of the hamstring muscles on the posterior portion of the capsule, thus relaxing the latt
tissues of the joint.
placing pillows under
bend the knees while lying on the side.[25] As the disease progresses further flexion is caused by muscular spasm. Because of this continued flexion of the knee, actual shortening occurs in the hamstring muscles so that the weight of the leg rests upon only the heel and the hip. This is due, at least in part, to contracture of the hamstrings. Outward rotation of the tibia on the femur usually follows within a short space of time.[36] The prevention of such flexion deformity of the knee rests in general with the practitioner.

When correction of flexion deformity at the knee has been attained by serial plaster casts and one is ready to begin weight bearing, support and protection must be given because of the weakened quadriceps muscles In

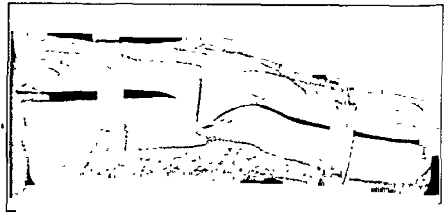


FIG 117 —Plaster cylinder, bivalved extending from upper thigh to lower third of leg This permits walking with a straight leg when the muscles are weak.

severe cases, it may be necessary to use metal leg caliper braces with leather
... into appropriate sockets
... ices of
... til the
quadriceps muscles have become stronger. If the quadriceps muscles are strong, one may employ a laced elastic knee support with or without metal
... on
... id
... s
... ie
... t,
feet are the stiff, pronated or flat feet and ...
if not all, of the disability of the feet can be prevented by proper early treatment. With the occurrence of an arthritis involving the feet, there results a limitation of motion in the tarsal and phalangeal joints, accompanied by muscular weakness. The main factor in the progression of deformity in the feet is this muscular weakness and the added burden of weight bearing. In

Castex Splints.—Castex splints have been used by some physicians. This material utilizes a synthetic resin. Bandages of this material (moist-

Application of Plaster Splints
A. If one desires to splint the knee joint temporarily a simple method for the general use of the practitioner is as follows.
1. The patient lies on his abdomen.
palm of the hand.
5. The plaster is allowed to dry.
6 The splint can then be lifted away easily because of the petroleum jelly coating
7. The splint may be placed on a radiator to dry more quickly. Splints may be made much lighter than usual if these are allowed to harden completely before using
8 Line the splint with felt or sheet cotton and apply to the limb with a bandage.
4 Plaster strips may be purchased
5.
and is then fastened to the leg with a bandage. Such splints are only for temporary wear They are much less effective than circular casts.
C The technic, as described for splinting the knee joint, may also be used to make a molded posterior body shell When a large shell is to be made, numbers of criss-cross plaster strips should be placed, especially about the hips and buttocks, where strain is to be expected Such splints are well molded about the hips and lower ribs are well padded
D
of the splint.

bed an equinus (plantar flexion) deformity will develop in arthritis unless a plaster cast is worn, or in the milder cases, a cradle or foot board is used.

Deformities at the hip or knee may also result in foot strain and deformity. Continued flexion of the knee may cause strain and acute pain in [illegible]

when weight bearing is resumed.

Deformities seen in the arthritic foot include: (1) rigid valgus, (2) equinus and (3) flat anterior arch with cocked up toes. The position of equinus or *plantar flexion results from the pull of the strong gastrocnemius muscle,* from the weight of the bed clothing and, in part, from gravity. When such patients are allowed to walk, this deformity is aggravated by the fact that

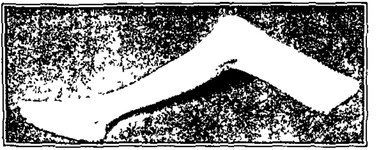


FIG. 118.—Foot drop with deformities at the knee, ankle and toes in neglected arthritis.

walking is frequently done mainly on the anterior part of the foot (because [illegible]

is also common. Subluxation and distortion at the metatarsophalangeal joints are common.

Pain in the foot in arthritis may be due to: (1) localized pressure, (2) *muscular or ligamentous strain;* or (3) acute or chronic inflammatory changes. The pain may be relieved temporarily by support and later by restoration and maintenance of normal function of the foot.

Pain due to excessive pressure may result from irregularities of the foot pressing against a smooth shoe or from an undue amount of weight being placed on certain bones of the foot. The commonest cause of pain in the feet and legs is foot strain, a result of long hours of standing, excessive weight and faulty body mechanics. The foot pain may be the result of an actual inflammatory process occurring in the joints of the feet or along the ligaments or tendon sheaths.

One of the most frequent causes of deformity of the foot in the arthritic and in other individuals is rest in bed over long periods of time with the bed clothes tightly drawn so that the feet are pulled into a position of equinus (plantar flexion). In any patient where rest in bed is required, it should be a rule that the bed clothes be placed loosely over the feet, or if this cannot be done, that a cradle be placed over the feet so that the bed

employs a soaking time of about a minute in water at a temperature of from 70° to 95° F.

If one desires to reinforce casts that encase the knee, elbow, wrist or ankle, the reinforcing splints will contribute more to the strength of the cast if they are narrow and thick, these splints should be placed on the front and back (and not on the sides) when being incorporated into plaster casts. Reinforcement for hip spicas should be placed with the strongest reinforcement laterally but with reinforcement also anteriorly and posteriorly; reinforcement in shoulder spicas should be over the top of the shoulder and in the axilla.

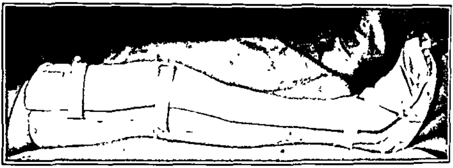


FIG. 115.—Resting leg cast cut into two halves to facilitate removal. This should extend from the upper thigh to the toes

[illegible]

weeks. When one employs cellulose acetate or a solution of celluloid in acetone to coat the splint, one can make lighter splints because these substances give the splint greater strength. Quickly drying paint can also be used.

Celluloid or Cellulose Acetate Splints.—If such light splints are desired, they can be made as follows: The part to be splinted is coated with petroleum jelly. Layers of plaster are applied over this forming a light plaster casing. When this has hardened the plaster is cut and removed. This mold is then filled with a mixture of plaster and water, or plaster, sawdust and water so that a cast of the leg is made within the mold. When this has hardened, the outer mold is removed and one is left with a cast of the part [illegible] the plaster cast of the part and this

[illegible]

made celluloid jacket or leg cast. Because of the resiliency of the material, [illegible] the part at will

[illegible]

celluloid jacket or leg cast may be laced as desired.

knee is the most comfortable position when swelling is present as it lessens the pull of the hamstring muscles on the posterior portion of the capsule, thus relaxing the latter, and permitting additional room for the swollen tissues of the joint. Most of these patients aid the flexion deformity by placing pillows under the knees to relieve discomfort. Other individuals bend the knees while lying on the side.[25] As the disease progresses further flexion is caused by muscular spasm. Because of this continued flexion of the knee, actual shortening occurs in the hamstring muscles so that the weight of the leg rests upon only the heel and the hip. This is due, at least in part, to contracture of the hamstrings. Outward rotation of the tibia on the femur usually follows within a short space of time.[36] The prevention of such flexion deformity of the knee rests in general with the practitioner.

When correction of flexion deformity at the knee has been attained by serial plaster casts and one is ready to begin weight bearing, support and protection must be given because of the weakened quadriceps muscles In

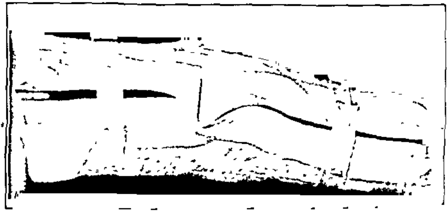


FIG. 117 —Plaster cylinder, bivalved extending from upper thigh to lower third of leg. This permits walking with a straight leg when the muscles are weak.

severe cases, it may be necessary to use metal leg caliper braces with leather ... sockets

... ces of

... til the

quadriceps muscles have become stronger. If the quadriceps muscles are ... without metal

pr ...

feet are the stiff, pronated or flat feet and deformities of the toes. Most, if not all, of the disability of the feet can be prevented by proper early treatment. With the occurrence of an arthritis involving the feet, there results a *limitation of motion in the tarsal and phalangeal joints,* accompanied by muscular weakness. The main factor in the progression of deformity in the feet is muscular weakness and the added burden of weight bearing. In

Castex Splints.—Castex splints have been used by some physicians.

Application of Plaster Splints

plaster are used, these may be cut into strips of the desired length, or rolled onto the leg wet.

4. The plaster is molded to conform to the shape of the leg with the palm of the hand
5. The plaster is allowed to dry
6. The splint can then be lifted away easily because of the petroleum jelly coating
7. The splint may be placed on a radiator to dry more quickly. Splints may be made much lighter than usual if these are allowed to harden completely before using
8. Line the splint with felt or sheet cotton and apply to the limb with a bandage.

B. Splinting of the lower leg and ankle
1. The patient should lie prone (on his abdomen).
2.
3.
4. Plaster strips may be purchased in varying sizes, or the physician may cut these from the rolls of plaster. If he desires, he may form his plaster splint by wetting the roll of plaster and rolling this back and forth on the back of the leg and sole of the foot
5. The plaster splint is removed when firm, padded on the inner side and is then fastened to the leg with a bandage. Such splints are only for temporary wear. They are much less effective than circular casts

C. The technic, as described for splinting the knee joint, may also be used to make a molded posterior body shell. When a large shell is to be made, numbers of criss-cross plaster strips should be placed, especially about the hips and buttocks, where strain is to be expected. Such splints are well molded about the hips and lower ribs are well padded.

D. For the hands and fingers, three to seven thicknesses of plaster gauze can be held against the hand and forearm which have been coated previously with petroleum jelly; these are held in the desired position until hardened. They are then lifted away and dried. *In making* plaster splints for the hands and fingers, the plaster is applied to the flexor aspect of the forearm and palm, usually leaving the thumb out of the splint

bed an equinus (plantar flexion) deformity will develop in arthritis unless a plaster cast is worn, or in the milder cases, a cradle or foot board is used.

Deformities at the hip or knee may also result in foot strain and deformity. Continued flexion of the knee may cause strain and acute pain in



FIG 118 —Foot drop with deformities at the knee, ankle and toes in neglected arthritis

walking is frequently done mainly on the anterior part of the foot (because this results in least jarring and consequently in less pain in the foot). Con-

joints are common

Pain in the foot in art[illegible]

muscular or ligamentous

changes The pain may

restoration and maintenance of normal function of the foot

Pain due to excessive pressure may result from irregularities of the foot pressing against a smooth shoe or from an undue amount of weight being

inflammatory process occurring in the joints of the feet or along the ligaments or tendon sheaths

One of the most frequent causes of deformity of the foot in the arthritic and in other individuals is rest in bed over long periods of time with the bed clothes tightly drawn so that the feet are pulled into a position of equinus (plantar flexion) In any patient where rest in bed is required, it should be a rule that the bed clothes be placed loosely over the feet, or if this cannot be done, that a cradle be placed over the feet so that the bed

Light Aluminum Splints.—Light aluminum splints can be used for the hand Sheet aluminum is employed 0.064 inch thick. This is rigid enough to maintain any desired shape and light enough not to be cumbersome. From this splints can be prepared promptly for many purposes.

Lucite Splints.—The du Pont preparation, Lucite, may be used as a splinting agent. Thin strips of this colorless, smooth substance may be softened in warm water and quickly molded to the affected part; the substance hardens readily on cooling. The material is very light, clean and possesses

Gelati

tional fa

already

low grade inflammatory processes in the legs. They are sometimes of value in arthritis of the foot with persistent edema.

RELIEF OF MUSCLE SPASM

While the earlier reports on the use of prostigmin[43] were optimistic, *later controlled studies showed very little if any relief of muscular spasm in rheumatoid arthritis by the use of prostigmin* Our personal experience with the use

to insure slow absorption (since more than a lethal dose of curare is given)[44] Curare in the limited period in which it has been used has seemed to be somewhat more effective than prostigmin, but it does not relieve muscular spasm effectively.

The final word on the usefulness and indications of these substances has not yet been said At the present time we can only say that they do not replace splints in the treatment of rheumatoid arthritis. Additional drugs, such as intravenous procaine injections,[13] and Tolserol (Myanesin) are being tried for possible relaxing effects—so far not too successfully.

is a time-

flamed

trial

before resorting to drugs.

PREVENTION OF DEFORMITY

It is usually much easier to prevent a deformity in arthritis than to cor-

sually from

inarily in a

in the development of deformities include pressure of the bed clothes, sagging bed, muscular strain, static influences and gravity.[17] Several pillows under the head may force the cervical and dorsal spine into forward flexion of the thoracic spine, flexion of the hip with contraction and shorten-

knee is the most comfortable position when swelling is present as it lessens the pull of the hamstring muscles on the posterior portion of the capsule, thus relaxing the latter, and permitting additional room for the swollen tissues of the joint. Most of these patients aid the flexion deformity by placing pillows under the knees to relieve discomfort. Other individuals bend the knees while lying on the side.[25] As the disease progresses further flexion is caused by muscular spasm. Because of this continued flexion of

flexion deformity of the knee rests in general with the practitioner.

When correction of flexion deformity at the knee has been attained by serial plaster casts and one is ready to begin weight bearing, support and protection must be given because of the weakened quadriceps muscles In

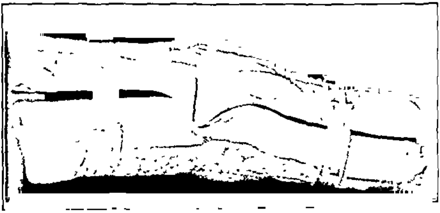


FIG 117 —Plaster cylinder, bivalved extending from upper thigh to lower third of leg This permits walking with a straight leg when the muscles are weak.

... with leather

quadriceps muscles have become stronger. If the quadriceps muscles are

limitation of motion in the tarsal and phalangeal joints, accompanied by muscular weakness The main factor in the progression of deformity in the feet is this muscular weakness and the added burden of weight bearing. In

ing of the powerful ilio-psoas muscles, flexion and external rotation of the knees.[41]

Resting in a chair for long periods is not advisable for arthritic patients as the position assumed leads to flexion contractures of the spine, hips, knees, elbows and wrists with internal rotation and adduction of the shoulders and pronation of the forearms.[18]

One of the first principles in preventing deformity is to relieve muscular spasm.[16] In cases in which there is a possibility of ankylosis, the parts should be maintained in positions which will give the patient maximum function.[20]

Deformities can be prevented if the physician has an understanding of the problem at hand and if the patient is cooperative.

The Knee.—The knee is the largest joint in the body and is frequently attacked in arthritis. Atrophy of the muscles about the joint may appear

Positions of Optimum Function	
Knee	A few degrees of flexion (10 to 15 degrees—no more)
Ankle	The foot should be at a right angle; very slight pronation is also sometimes advisable
Hip	Slight flexion of the hip with the leg practically straight, no rotation, abduction or adduction
Shoulder	Not more than 45 degrees between the humerus and the posterior border of the scapula, forward flexion (30 to 45 degrees) and rotation of the arm in slight supination (10 to 15 degrees), so that the hand will reach the mouth.
Elbow	Depends upon the patient's occupation and wishes, in general, approximately 70 to 80 degrees of flexion is usually best with forearm in slight supination (10 to 15 degrees)
Wrist	About 35 degrees of dorsiflexion (i.e. extension)
Hand	30 degrees of flexion of the fingers and metacarpophalangeal joints, slight abduction of the thumb
Jaws	Open 1 inch at least
Spine and Head:	In good posture, head straight forward

rapidly. Common findings include narrowing of the joint space, fixation of the patella against the femoral condyles (often with fusion), subluxation of the tibia on the femur, and contracture of the joint capsule, hamstring muscles and periarticular structures.

In acute arthritis involving the knee, the patient should be put to bed. No weight bearing should be permitted.[42] If there is inability to straighten the knee a plaster cast should be applied from toes to groin with the foot at right angles and the knee as fully extended as possible. As pain and muscular spasm subside a new plaster cast is applied and further casts are used until the knee can be fully extended. A plaster cast is worn day and night in acute cases,[F] but is removed at least once every twenty-four hours to exercise and to permit the use of heat which aids in lessening pain and muscular spasm.

the medial aspect of the knee and the anterior thigh In these cases, examination may disclose some muscular spasm after exercise, limitation of rotation and abduction, and flexion deformity.



FIG 119 —Short leg cast to prevent and correct deformities of the foot Note the foot at right angles and slight varus The arches of the foot have been molded in the plaster

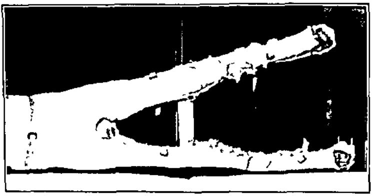


FIG 120 —Hip spica, including both hips, with reinforcing bar at knees The spica has been cut into an anterior and posterior half

Deformities of the hips and spine go hand in hand Flexion of the hips follows distortion of the trunk in order to maintain balance. Flexion deformities of the hip result in forward tilting of the pelvis with further compensatory lumbar lordosis.

Light Aluminum Splints.—Light aluminum splints can be used for the hand. Sheet aluminum is employed 0 064 inch thick. This is rigid enough to maintain any desired shape and light enough not to be cumbersome. From this splints can be prepared promptly for many purposes.

Lucite Splints.—The du Pont preparation, Lucite, may be used as a splinting agent. Thin strips of this colorless, smooth substance may be softened in warm water and quickly [illegible] stance hardens readily on cooling.

[illegible]

already prepared gelatin gauze bandages are often useful in phlebitis and low grade inflammatory processes in the legs. They are sometimes of value in arthritis of the foot with persistent edema.

RELIEF OF MUSCLE SPASM

While the earlier reports on the use of prostigmin[13] were optimistic, *later controlled studies showed very little if any relief of muscular spasm in rheumatoid arthritis by the use of prostigmin.* Our personal experience with the use [illegible]

to insure slow absorption (since more than a lethal dose of curare is given)[14] Curare in the limited period in which it has been used has seemed to be somewhat more effective than prostigmin, but it does not relieve muscular spasm effectively.

The final word on the usefulness and indications of these substances has not yet been said At the present time we can only say that they do not replace splints in the treatment of rheumatoid arthritis. Additional drugs, such as intravenous procaine injections,[13] and Tolserol (Myanesin) are being tried for possible relaxing effects—so far not too successfully.

[illegible]

before resorting to drugs.

PREVENTION OF DEFORMITY

It is usually much easier to prevent a deformity in arthritis than to correct one *Deformities in most arthritic patients can be prevented.*

During the early stages of an arthritis, deformities result usually from muscular spasm with joint irritation Such deformity is ordinarily in a position of flexion and often adduction, because this affords the patient the greatest relief from pain and because of the more powerful action of most flexor and adductor groups of muscles over the corresponding extensor and abductor groups Other factors which favor muscular contraction and aid in the development of deformities include pressure of the bed clothes, a sagging bed, muscular strain, static influences and gravity.[17] Several pillows under the head may force the cervical and dorsal spine into forward flexion of the thoracic spine, flexion of the hip with contraction and shorten-

clothes rest upon this or upon a pillow placed under the covers at the foot of the bed rather than the feet; otherwise, lying in bed leads to external rotation of the hips, slight flexion of the knees and mild equinus of the feet.

[illegible]

If any tendency to foot drop is seen, support the foot in a position at right angles to the leg by molded splints (to be removed daily for motion of the foot)

In arthritis involving the foot, **permit no weight bearing until pain and swelling have completely disappeared**, and until the muscular tone of the foot muscles has been re-educated

Apply hot fomentations or other forms of heat to the foot and ankle

Prevent muscular atrophy in the foot by exercises while the patient is confined to bed.

[illegible] plates if necessary.

[illegible]

[illegible] *correct* them

Employ proper physical therapeutic procedures, including contrast baths after the acute stage of the arthritic process has subsided.

If there is any tendency to foot drop, the foot should be held at right angles by a posterior molded splint or cast (removed once or twice a day to permit free motion of the foot), or the head of the bed may be elevated 6 inches allowing the body to slide down somewhat in bed so that the feet may press against an upright foot board.

With the onset of rheumatoid arthritis in the ankle, muscular spasm *tends to produce a valgus position of the foot*, while the pull of the gastrocnemius muscle, the weight of the bed clothes and gravity lead to an equinus position of the foot. The equinovalgus which occurs leads to depression of the metatarsal arch. With weight bearing a spread of the forefoot follows. Later there is cocking up of the toes and in some instances hammer toes.

When the feet are involved in an arthritic process, no weight bearing should be permitted until pain and swelling of the feet have entirely subsided, and until the foot muscles have been strengthened by non-weight-bearing exercises. *The muscles of the feet become weak and atrophied with prolonged rest in bed from any cause, unless active movements have been carried on throughout the illness.* The patient must have proper fitting shoes and proper support to the arches of the foot before attempting to walk again

If these procedures are carried out fully, the late changes in arthritic feet, particularly the peroneal spastic flat foot, seen so frequently in ambulatory patients, can be prevented.

Prevention of Flexion Deformity of the Knees
Never place pillows under the knees for comfort in these patients; this is the commonest cause of such a flexion deformity.[21]
Use a posterior molded splint or cast extending from the upper part of the thigh to the toes with the knee in full extension. A full cast including the foot is best.
Incorporate a wooden strip into the leg splint posteriorly at the ankle to control rotation of the hip joint and prevent outward rotation of the lower leg.
Remove the splint once daily and allow active movements.
Apply hot fomentations or other heat to the knee twenty to thirty minutes several times a day.
Employ muscle setting and non-weight-bearing exercises until the acute inflammation has subsided.[8]
With improvement, lessen the period of splinting gradually so that within a short time, splinting may be required only at night, until inflammation has subsided.
Even in the later stages of the process, permit the splints to remain in place all night as flexion of the knees is common during sleep (unless prevented by splints).
[illegible]
[illegible] tally while sitting. During the first few weeks or months of walking, it may be advisable to use a stiff-legged brace, an elastic (Ace) bandage or a cane. A metal or wooden walker is of great assistance to these [illegible]
[illegible]
Massage should not take the place of exercise.[22]

One of the fears of the patient with acute joint involvement is excruciating pain which may occur on slight motion of the joint. With the joint held firmly in a bivalved cast painful movement of the joint does not occur. If all movement is painful, muscle-setting exercises can be taught. These can be carried out without motion in the joint. No passive motion should be done. Following this the splints are reapplied. As long as joint pain, tenderness and swelling persist, it is advisable that splints be used for the affected joint during most of the day and night so that muscular spasm will not cause deformities.[23]

The commonest *deformities of the knee are flexion, subluxation and outward rotation.* The cause of this flexion deformity is the desire of the patient to relieve the pain in the acute stage of the disease. Flexion of the

often secondary to tears, hemorrhage or calcification in the supraspinatus tendon. Periarthritis and fibrositis are also common.

Prevention of Deformity of the Shoulder in Arthritis.[6]—In an effort to [illegible]

thirty minutes several times a day during the acute stage of an arthritis to relax muscular spasm and relieve discomfort

---

***Prevention of Deformity of the Hip Joint in Arthritis*[10]**

Rest in a firm, flat bed

If permanent hip flexion is to be prevented or avoided, *the patient should lie on his abdomen* (prone) *for thirty minutes several times a day, beginning with one or two pillows placed under the abdomen* The pillows are gradually removed as tolerated permitting the stretching of the anterior structures which will allow the hip to assume a neutral position If the flexion deformity is slight, it may be corrected by traction in the line of the deformity, together with hot fomentations for thirty minutes two or three times a day

If there is a tendency to rotation and adduction deformity, hold the lower extremities mid-way between external and internal rotation, and, at [illegible]

with a cross-bar under the ankle, to prevent rotation

In acute painful hip joints, it may be necessary to apply a bilateral plaster spica in order to secure relief or to correct deformities The [illegible]

Frequently change the patient's position The bed must have a firm [illegible]

function

Proper exercises should follow the subsidence of the acute process Prone exercises to strengthen the gluteal muscles should be given

Permit no weight bearing until pain, tenderness and muscle spasm have subsided

When weight bearing is resumed, a walker, and later crutches, should be used to prevent irritation and to lessen the weight-bearing load upon the diseased hip joint This should be continued until muscular spasm subsides

---

Prevention of Deformities in Arthritis
Physiologic rest of the involved joints in the acute stage by. Rest in a firm bed. Splinting of the affected joints (by the use of casts, splints or other apparatus) to prevent trauma and allay muscular spasm. Active movement of all joints through as wide a range of painless motion as possible several times a day. Special graded muscular exercises for each joint involved, with periods of rest following such exercise Maintenance of good posture (see section on Orthopedics).

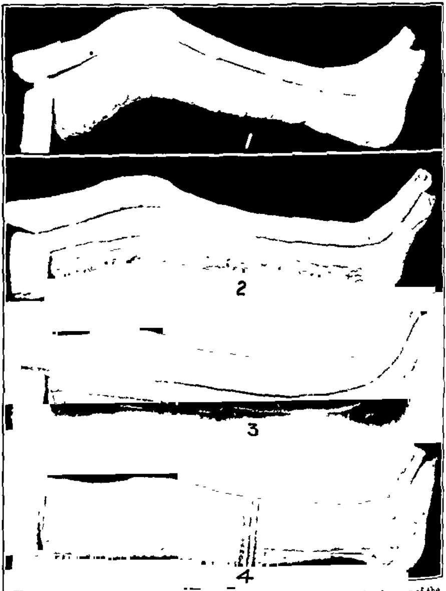


Fig. 116.—Gradual correction of flexion deformity of the knee by weekly changes of the plaster cast.

rect flexion at the hip. This plaster spica is bivalved and removed several times a day for heat and exercises.

The Spine.—If the cervical spine is involved, the tendency toward a flexion deformity (with at times rotation of the head) may be overcome by using *only one small pillow* under the head, and by lying flat in bed without

traction at the head of the bed.

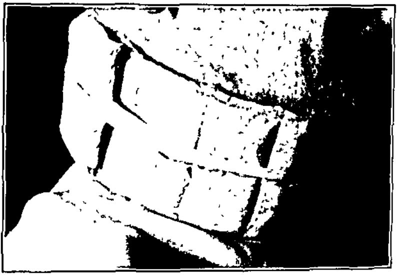


FIG 121 —Padded collar worn for spasm and deformity in the cervical spine

Deformity may be prevented by a Thomas collar, or by a piece of felt wrapped around the neck each night. The latter should be about ⅜ inch

width at one

e other.

ow end,

um sized

be wrapped about the neck in a

uscular spasm, one can employ

sand bags, hot fomentations, traction or plaster collars for support and

shoulder support

'bowing

ribs

rthritis,

but to subacromial bursitis or involvement of the shoulder cuff area (which includes the muscular attachments to the tuberosities). The *bursitis* is

To Avoid Deformities of the Thoracic and Lumbar Spine [19]
1 Lie on a firm bed with only a small pillow or no pillow. Lie on the back or abdomen most of the time
2 The patient should lie on the back for thirty minutes after each meal, with the hands beneath the head A pillow should be placed under the knees to flatten the lumbar spine Later a small pillow may be used under the thoracic spine to combat forward bowing of the spine
3 In very painful cases or those with marked muscular spasm a plaster shell is molded over the back The patient lies in this most of the time
4 Breathing exercises, back stretching exercises, and later postural exercises are performed 2 or 3 times daily These should not cause pain
5 As soon as muscular spasm has subsided and the flexion deformity of the spine has decreased, a plaster jacket is applied over Goldthwait irons In this the patient may be ambulatory Another jacket is applied as soon as further correction of the spinal deformity is possible
6 A spinal brace with shoulder straps may be used when the patient shows no further tendency to forward flexion of the spine Eventually no support is required

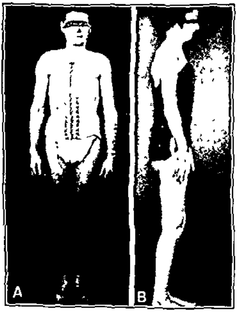


FIG 124 —*A* Celastic jacket The jacket is cut low in back and extends to the sternal notch in front This prevents forward bowing of the spine Lacings or straps can be used after the jacket is cut in front for removal *B* Lateral view of leather jacket

16 Henderson, M S · Arch Physiotherapy, 17, 563, 1936
[illegible]
[illegible]
35 Small, J C Am Heart Jour, 37, 1090, 1949.
36 Stump, J P: New York State Jour. Med, Dec 1938
37 Swaim, L. T.: Jour Indiana Med Assn, 38, 487, 1945
38 ——— Jour Bone and Joint Surg, 18, 80, 1936
39 ——— Am Jour Nursing, 36, 211, 1936
40 Swaim, L T, and Kuhns, J G Jour Am Med Assn, 93, 1843, 1929
41 Thorndike, A, Jr, and Garrey, W E. New England Jour Med, 218, 203, 1938
42 Todd, A H Lancet, 1, 515, 1922
43 Trommer, P R., and Cohen, A Jour Am Med Assn, 124, 1237, 1944

When the patient is ambulatory, a platform splint may be used to maintain the abducted position. Splints must be removed daily and motion

... [26]

... y inserting

... tolerated,

perform exercises to maintain abduction and external rotation. Employ



FIG. 122 —Head traction The patient lies flat in the bed. Traction (5 to 10 pounds) is applied through a Sayre head sling attached to weight at the head of the bed.

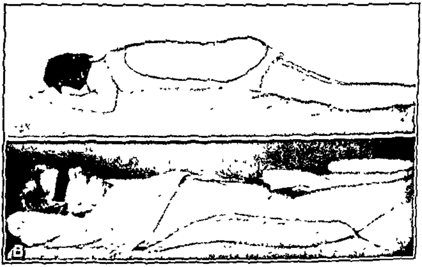


FIG. 123 —Plaster shell for the back. *A*. This is made with the patient lying prone on a thick pillow *B*. The patient lying in the shell

more vigorous physical therapy following subsidence of the acute process When weakness or deformity has been present a long time, elastic shoulder straps attached to a brace or corset give considerable support and relief of muscular strain.[24]

... screw eyes

The ends

...ne end of

CHAPTER 20

CORRECTION OF ARTHRITIC DEFORMITIES

By John G. Kuhns, M.D. and Theodore A. Potter, M.D

In *severe rheumatoid arthritis deformities often develop in spite of the* measures outlined in the previous chapter. Many times, also, the physician is faced with a patient who has already developed articular deformities before seeking his treatment If these *deformities are mild or early*, the employment of the measures previously outlined, such as application of traction, use of rest splints, or even serial application of plaster casts in position of maximum extension can correct the deformity and no more elaborate or radical procedure is needed

When serial changes of plaster casts, traction, or corrective exercises fail to correct deformities, the following measures may be considered skeletal traction, turnbuckles attached to Kirschner wires, manipulation of the joint under anesthesia, surgery of the juxta-articular connective tissues, or surgery of the bones and joints. The above procedures should not be carried out in the presence of active joint inflammation The only exception to this rule is in the presence of severe deformity which prevents adequate nursing care. In rheumatoid spondylitis, for example, severe adduction deformity of both hips occasionally requires prompt manipulation of the hips into abduction, with application of a bilateral plaster spica, in order to permit proper nursing care. (Fig. 120.)

Skeletal traction is employed chiefly in the lower extremity for the purpose of correcting flexion deformities at the hip and knee and subluxation at the knee Traction of more than 15 pounds can rarely be employed The bones are weak and the Kirschner wire will pull through the bone if too great traction is used. Skeletal traction must be supervised constantly to be effective It is much less comfortable than other equally effective measures For these reasons it is not often used in the treatment of chronic arthritis (See Fig 131.)

Turnbuckles attached at the sides of the deformed joint to Kirschner *wires drilled through the bone and attached to stirrups above and below* the joint are a simple and effective means of correcting subluxation and flexion deformities. While this method can be used in all of the major joints, it is used most commonly about the knee The limb is kept in balanced *Russell's suspension while the stretching is progressing* The turnbuckles separate the bone ends by one or two turns each day. At this rate no great pain is felt. The deformity is usually corrected in one to two

to four days when physiotherapy and exercises are begun. [illegible] usually shows lateral instability for one to two months but no persistent instability has been observed in any case.

To Avoid Deformities of the Thoracic and Lumbar Spine [22]

1. Lie on a firm bed with only a small pillow or no pillow Lie on the back or abdomen most of the time
2. The patient should lie on the back for thirty minutes after each meal, with the hands beneath the head A pillow should be placed under the knees to flatten the lumbar spine Later a small pillow may be used under the thoracic spine to combat forward bowing of the spine.
3. In very painful cases or those with marked muscular spasm a plaster shell is molded over the back The patient lies in this most of the time.
4. Breathing exercises, back stretching exercises, and later postural exercises are performed 2 or 3 times daily. These should not cause pain
5. As soon as muscular spasm has subsided and the flexion deformity of the spine has decreased, a plaster jacket is applied over Goldthwait irons In this the patient may be ambulatory Another jacket is applied as soon as further correction of the spinal deformity is possible
6. A spinal brace with shoulder straps may be used when the patient shows no further tendency to forward flexion of the spine Eventually no support is required

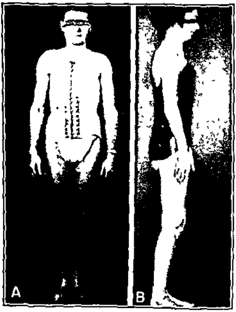
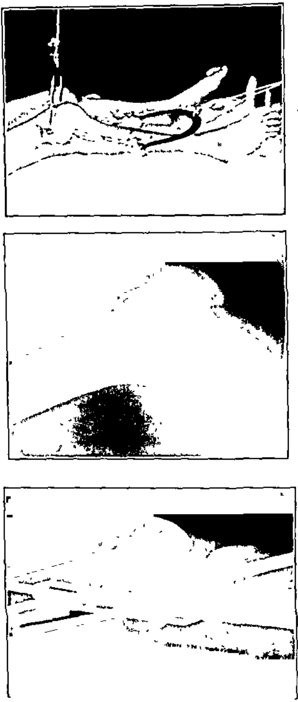


FIG 124 —*A* Celastic jacket. The jacket is cut low in back and extends to the sternal notch in front This prevents forward bowing of the spine Lacings or straps can be used after the jacket is cut in front for removal *B* Lateral view of leather jacket



A

B

C

FIG. 131.—A. Skeletal traction with Kirschner wire applied to the knee joint. B. X-ray of knee with flexion and subluxation before traction was applied. C. X-ray of knee after removal of traction. The flexion and subluxation have been corrected.

the rope, the other arm is pulled up and vice versa. In chronic arthritis of the shoulders, the patient should attempt to clasp the hands behind the head and flatten out the flexed elbows on the bed for periods of ten minutes three times a day. The reader should refer to the chapter on "The Painful Shoulder" for a more detailed discussion of this subject, p. 958.

The common deformities occurring in arthritis involving the shoulder joint are adduction and internal rotation of the arm; true ankylosis of the shoulder joint rarely occurs in arthritis. We have seen only one ankylosis of the shoulder in over 5000 patients suffering from rheumatoid arthritis

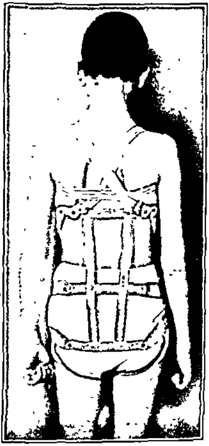


FIG 125 —Spinal brace with elastic shoulder straps. This is more comfortable but gives less rigid support than a plaster jacket

The Elbow.—The first deformity is slight flexion followed by limitation

Manipulation.[51]—*This is the forceful stretching of a joint under anesthesia.* Manipulations are carried out for the relief of two pathological conditions: adhesions within and about the joint and contractures in the muscles and fascia. Often these can be prevented from developing or the milder ones can be corrected by casts and by exercises. If casts have been used conscientiously for several weeks without any improvement in the deformity, manipulation of the joint should be considered. This should not be done if there is acute inflammation in the joint, if the individual has fever, if x-rays show ankylosis, or if the bones and muscles are markedly atrophied

As a rule, a general anesthesia is given; spinal anesthesia or brachial plexus block is occasionally used. Since the procedure is usually of short duration nitrous oxide or intravenous sodium pentathol are the anesthetics of choice. When the pa
limb on the distal side n
firmly by an assistant



FIG. 132.—Manipulation of the knee under anesthesia. The assistant holds the bone just above the joint The operator manipulates gently with short leverage

slowly and forcibly extended. The joint should be stretched only once, the limb should not be "pump-handled" With short leverage there is much less danger of fracturing the atrophic bones (a real danger in every manipulation in arthritis) There is also less danger of tearing blood vessels and nerves. Cracking noises are always heard as adhesions are freed The stretching of contractures makes no sound. If the surgeon fears that further stretching may fracture bone or tear tissues, he should stop A further manipulation can be undertaken in one to two weeks [52]

After manipulation the joint is held in the improved position with a plaster cast until soreness about the joint subsides. Then physiotherapy and exercises are given. A few gentle manipulations may be required to correct a deformity completely. If no improvement follows one manipulation, further manipulations will also be useless and should not be under-

The elbow joint is often sprained by arthritic patients. It is a non-weight-bearing joint, but the arthritic individual supports himself by holding on to chairs or tables, or by using a cane if the legs are weak or deformed thus employing the elbow as a partial weight-bearing structure. This additional trauma to the elbow joint is of importance in leading to further destruction of the joint. The customary attitude assumed by the arthritic patient in bed with the elbow at an angle of 90 degrees or less and the hand in pronation on the chest assures the patient of comfort but encourages a flexion deformity

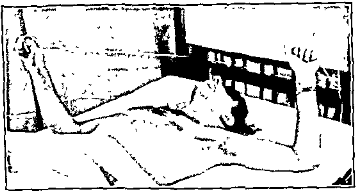


Fig 126 —Rope and pulley for shoulder exercise

Prevention of Deformity at Elbow

Heat may be used to lessen muscular spasm and pain at any stage of the arthritis Hot moist compresses wrapped around the elbow are best
The commonest deformity at this joint is flexion with pronation of the forearm [31] Prevent such deformity by.
In acute arthritis of the elbow, a splint should be worn with the elbow at 70° including the wrist The splint is removed at least once daily to permit several active movements of the joint

hinges can be worn

The Wrist and Hand.—Palmar flexion is the common deformity of the wrist [27] Deformity is due to the pull of the strong flexor muscles and to gravity The flexion deformity which results decreases the normal grasping power of the hand, because the fulcrum of the flexor tendons is changed. In acute arthritis the ligaments about the distal end of the ulna become stretched by the swelling and become relaxed Dorsal subluxation of the ulna is common This often becomes permanent. In late arthritis the wrist may become fused in flexion and ulnar deviation The lower

suspension into the joint space just before or after manipulation, has been found to prevent the increased pain and reflex muscle spasm which may follow manipulation.[35] Manipulation here should be followed by exercises to keep a full range of motion at the shoulder. At the hip joint the ability to cross the knees and to tie the shoes can often be regained by a gentle manipulation of the hip in abduction and internal rotation. In these joints the greater range of motion gained is again lost in a few months if the patient does not continue to perform daily stretching exercises. Manipulation has not been found helpful in other joints in osteoarthritis. In the presence of large spurs or extensive bony deformity manipulation is likely to cause harm.[13]

Manipulation of Deformed Joints:

- Should not be done during the active stage of an arthritis, when the patient is seriously ill, if x-rays show bony ankylosis or if there is severe atrophy of bones or muscles.
- Indicated in selected cases of deformity due mainly to adhesions within or about the joints or to muscular contractures.
- In degenerative joint disease rarely produces permanent improvement in range of motion or relief of pain, often helpful temporarily in milder cases.
- May be preceded by injection of 1 per cent procaine intra-articularly, or by 25 mg. of hydrocortisone acetate suspension, to minimize post-manipulation exacerbation of pain and spasm.
- Always performed under regional or general anesthesia, muscular relaxation is essential for success.
- Move the limb slowly, use short leverage, stretch the limb one time only.
- Great care should be taken during the manipulation because of the possibility of producing fractures of the atrophied bones or tears of the blood vessels or nerves.
- After manipulation apply a plaster cast with the joint in the improved [illegible]

ORTHOPEDIC SURGERY IN ARTHRITIS

The surgery of chronic arthritis is patchwork surgery.[58] If the arthritic process has been quiescent for six months and if the patient is gaining weight

radio-ulnar joint is involved in the fusion, preventing supination and pronation.

on the outstretched hand as in getting up from a chair or bed, and leads to the "flat hand deformity of arthritis."[14] In long-standing arthritis the carpal and metacarpal bones may become a single fused mass.

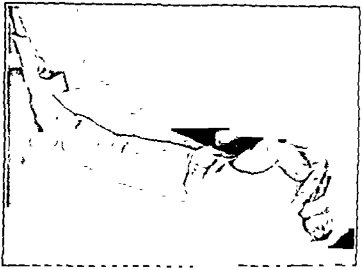


FIG 127 —Resting shell for the elbow The elbow is near right angle flexion The forearm is at neutral rotation

...rpophalangeal joints in ...xtensive and stretch the ...ts. There is a gradual flexion in these joints. At the metacarpo-phalangeal joints flexion is followed by ulnar deviation and subluxation. At the thumb flexion

the same plane as the fingers and opposition of the thumb becomes lost.[3]

Prevention of Deformities at Wrist and Hand

A cock-up splint from the upper forearm to the fingertips is applied to hold the wrist in 35 degrees of dorsiflexion and the fingers in 10° flexion, ulnar deviation must be prevented by a flange on the ulnar side.

Special exercises and occupational therapy for the hands are needed

and strength, operations on arthritic patients result in no greater likelihood of infection, ankylosis or lack of union than in other diseases. To determine the quiescence of the disease one should use an objective formula, e.g., that proposed by Steinbrocker,[71] and accepted as therapeutic criteria by the American Rheumatism Association.

Osgood[62] has stressed certain conditions which must be met before surgery is even considered in arthritic patients: Quiescence of the arthritic process for months. Optimal general condition of the patient. Good morale of the patient. Adequate facilities for hospital and follow-up care.

The importance of intensive training in muscular exercise and control for a period of at least several weeks prior to the operation is important. The patient is taught preoperatively to perform those exercises which will be rec[illegible]

[illegible] Wilson[illegible]

ha[illegible]

1. Is the arthritis still active?
2. Is the patient in good enough physical condition to permit extensive surgical procedures?
3. Considering the age of the patient, his physical state and the number of operations required, is the expected gain in function from operation worth the effort?
4. Should the operation be delayed for any reason for institution of other therapy?
5. Will the patient be able to cooperate in after care with the proper morale and willingness to endure some pain?
6. Are the facilities necessary for successful treatment present in the hospital?
7. Will the patient be able to receive proper after care on leaving the hospital?
8. Is the patient's financial status such that he can go through the entire treatment or have the resources of the community been mobilized to care for such treatment?

Principles in Joint Surgery

Do not operate during the active stage of an arthritis, joint surgery should be attempted only after a period of at least six months of quiescence from joint activity.

Patients who have been bedridden for years are poor operative risks. Their tissues heal only slowly.

Attempt to obtain a functionally useful joint. Orthopedic surgical procedures upon the upper extremity are concerned with the use of the arms, particularly for eating, dressing and personal care, while the leg problem is concerned with the ability to stand and walk.

When both arms and legs are disabled, an attempt should be made to solve the arm problem before the leg condition.

Develop surrounding muscles by exercises for several weeks prior to operation.

thumb in abduction and in extension. When only one finger shows per-

for cleanliness. Where the joints are destroyed and will probably become

extension and the first metacarpo-phalangeal joint should be fixed also in full extension Fixed plaster splints are best when the arthritis shows

extensor tendons most commonly above or below the transverse carpal ligaments Such ganglia may become smaller and subside entirely. When they become large and disturb function they should be removed surgically In rheumatoid arthritis the tendon sheaths often become thickened and show increasing fluid content, much as the joint capsules Later the tendon sheaths tend to become narrowed in their lumina This is the same parti-

If surgery is done too early in the arthritic, it can do more harm than good [87] *Surgery should always be looked upon as an adjunct in the treatment of arthritis.* In quiescent or arrested cases, surgery may be required to increase the mobility of the body with a minimum expenditure of the patient's energy. Surgery deals with the mechanical factor mainly in the arthritic, with very few exceptions, it should be resorted to only in arrested cases

Ankylosis, per se, does not require surgery. If the ankylosis is in poor position resulting in increased muscular fatigue, one should then employ the simplest type of osteotomy to correct the deformity. Very little surgery will be required if proper preventive orthopedics has been used *early in the disease, since ankylosis, if it occurs, should be in a good position* for function

There is no hard and fast rule concerning surgery in the arthritic One should discover whether the deformity is due to contraction of tendons or capsules or whether there is an actual bony deformity. One will then decide in that given case whether tendon lengthening, capsulorrhaphy, osteotomy or another procedure is preferable. Usually unless there is bony ankylosis, plaster casts and manipulation are first tried to see whether they will bring any correction in the deformity. If the deformity is resistant, surgery is next considered.

Aspiration and Arthrotomy.—Aspiration for the removal of synovial fluid in a markedly swollen knee often relieves pain and may prevent persistent relaxation of the articular capsule It is only temporarily helpful. As a diagnostic procedure it is sometimes helpful. (See chapter on Synovial Fluid, p 48.) Injection of procain solution or other analgesic into or about the joint tissues will sometimes relieve pain but for a short period only [11] The aspiration of fluid and intra-articular injection of hydrocortisone gives more lasting symptomatic and objective relief. The technic for this procedure is described in a separate chapter

Arthrotomy (the surgical opening of a joint) is used for the removal of loosened or damaged tissue, to make a diagnosis or because it interferes

is employed when there is persistent effusion into the joint or locking of the joint. At operation a loose body, synovial fringes, osteophytes, or a degenerated cartilage may be found. After arthrotomy the joint is splinted with a plaster cast or a voluminous pressure dressing for three to five days Then exercises are given

Synovectomy, the partial or complete removal of the lining membrane of the joint is performed most often at the knee but sometimes at the elbow, *ankle and metacarpal joints.* [79] *Synovectomy is indicated when there is* persistent thickening of the synovial membrane which interferes with function Usually *x*-ray therapy is given first in an attempt to shrink the thickened membrane before considering synovectomy.

Synovectomy of the Knee.—Hyperplastic[13] synovial membrane, thickened and thrown into folds may cause disability even after the arthritis is inactive Such hyperplasia in the knee often causes chronic effusion,

sheath is opened longitudinally. This usually relieves the symptoms permanently. If arthritis has been present in the hand for a number of years, thickening and contracture sometimes occurs in the palmar fascia. This is indistinguishable clinically and microscopically from Dupuytren's contracture. In the early stages this thickening can be helped by splinting and physiotherapy. When the palmar fascia is markedly contracted, preventing full extension of the finger, the same surgical measures are employed as are used in other types of Dupuytren's contracture.

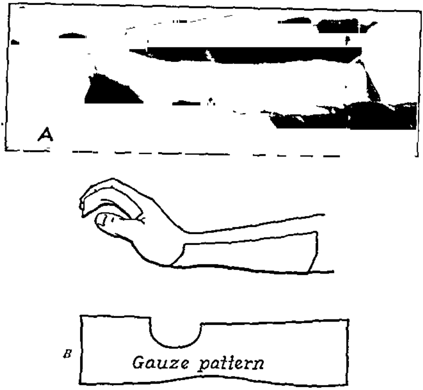


FIG. 128.—*A*. Resting hand cast. The fingers are slightly flexed. The wrist is dorsiflexed. The palmar arches are molded. *B*. Model for resting hand cast.

The Jaw.—Ankylosis of the temporomandibular joints rarely occurs in rheumatoid arthritis but deformity of the joint and malocclusion are common. In children with the juvenile form of rheumatoid arthritis, deformity of the lower jaw is common. The mandible is shorter than the maxilla and there is permanent malocclusion of the teeth.[33] In profile, the face shows the receding jaw, producing the so-called "bird-like facies."[8] (Fig. 130.)

In adults, malocclusion may occur from involvement of one temperomandibular joint. A more common deformity is a limitation of motion of the jaw, producing a narrowed bite. If these joints are involved, attempts to prevent ankylosis include the application of external heat for periods of fifteen to twenty minutes, 3 times a day, a soft or liquid diet, and the chewing of a large mass of gum several times each day.

the American Rheumatism Association.

Osgood[62] has stressed certain conditions which must be met before surgery is even considered in arthritic patients: Quiescence of the arthritic process for months. Optimal general condition of the patient. Good morale of the patient. Adequate facilities for hospital and follow-up care.

The importance of intensive training in muscular exercise and control for a period of at least several weeks prior to the operation is important. The patient is taught preoperatively to perform those exercises which will be

Wilson[63]

1 Is the arthritis still active?

2. Is the patient in good enough physical condition to permit extensive surgical procedures?

3 Considering the age of the patient, his physical state and the number of operations required, is the expected gain in function from operation worth the effort?

4. Should the operation be delayed for any reason for institution of other therapy?

5 Will the patient be able to cooperate in after care with the proper morale and willingness to endure some pain?

6. Are the facilities necessary for successful treatment present in the hospital?

7. Will the patient be able to receive proper after care on leaving the hospital?

8. Is the patient's financial status such that he can go through the entire treatment or have the resources of the community been mobilized to care for such treatment?

Principles in Joint Surgery

Do not operate during the active stage of an arthritis, joint surgery should be attempted only after a period of at least six months of quiescence from joint activity

Patients who have been bedridden for years are poor operative risks Their tissues heal only slowly

Attempt to obtain a functionally useful joint Orthopedic surgical procedures upon the upper extremity are concerned with the use of the arms, particularly for eating, dressing and personal care, while the leg problem is concerned with the ability to stand and walk

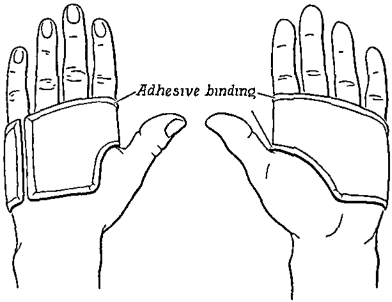
When both arms and legs are disabled, an attempt should be made to solve the arm problem before the leg condition

Develop surrounding muscles by exercises for several weeks prior to operation.

The temporomandibular joint may be splinted by an elastic strap under the chin, fastened to a cloth skull cap.[5] Wooden wedges (such as several tongue depressors taped together) or corks may be inserted at first between the incisor teeth and later moved back between the premolars to widen the bite. These should be inserted after the periods of chewing. Increased pain and stiffness within twenty-four hours following the chewing or wedging are indication that too rapid stretching has occurred.



A



B

Fig 129 —*A* Plaster cuff to prevent metacarpo-phalangeal dislocation and ulnar deviation of the fingers volar aspect *B* Diagram showing both dorsal and volar aspect

The sensory nerves supplying the articular capsule can be cut and in this way most of the pain from that joint can be relieved. Operations of this type are performed occasionally at the hip, knee and elbow. The most common of these is resection of the nerves going to the capsule of the hip, called obturator neurectomy.[61] The obturator nerve, or in exceptional cases the accessory obturator nerve, supplies the anterior capsule of the hip joint. Resection of the obturator nerve partially denervates the articular capsule of the hip. When there is pain both posteriorly and anteriorly the branches to the posterior capsule, accompanying the nerve to the quadratus femoris, are also cut. This procedure is simple, usually nonshocking and leads to no serious sensory or motor sequelæ. It is employed chiefly in elderly individuals when relief of pain is desired rather than improvement in function. Denervation has also been used to relieve pain in the elbow and knee. Nerve resection has not been performed often enough at the elbow or knee to give any definite indications for its use.

Operations on the Soft Tissues for Flexion Contractures.—These are required more frequently at the knee than at any other joint. A release of contracted soft tissue is sometimes required at the finger joints, elbow, hip and ankle joints. In all of these areas contraction of the articular capsule on the fle[illegible] capsule [illegible] their re-s[illegible] articular [illegible] these procedures are persistent flexion deformities which seriously interfere with function and which have not responded to splints or to manipulation.

At the interphalangeal joints, cutting the articular capsule after first [illegible]fficient to permit full [illegible] the biceps humerus [illegible] usually with severe flexion at the elbow joint extensive damage to the joint has occurred. A far more serviceable joint is obtained by performing an *arthroplasty.* Release of tight tendons and fascia is sometimes required about the hip. In flexion contracture the fascia lata must be cut. In severe flexion contractures the attachment of the rectus femoris tendon must be pushed off subperiosteally from the anterior superior and inferior iliac spines. In severe adduction contracture the tendons of the adductor longus and brevis must be cut at the pubic rami. Correction of adduction deformities can be accomplished by obturator neurectomies. At the ankle the tendo Achilles must be lengthened. After cutting soft parts, the joint is fixed in full extension until the tissues have healed in the lengthened position. Then exercises are given to strengthen the extensor muscles.

At the knee severe flexion contracture[54] and subluxation can often be corrected at one operation by a combined capsular release and tendon lengthening procedure which is called a *posterior capsuloplasty.* In this operation a lateral incision is made extending upward from the fibular head. The fascia lata with the intermuscular septum is cut, the tendon of the biceps femoris is lengthened. The capsule of the knee is freed from the posterior surface of the femur. In this way flexion deformity and subluxation can be corrected. The limb is then fixed in full extension in a plaster cast. Sometimes the contracture of the common peroneal nerve and popliteal artery are so severe that full extension would cause paralysis or

Acute involvement of the temporomandibular joint occurs in 20 per cent of patients with rheumatoid ar[illegible]
should be in centric occlusion,
even while eating or talking.[11] [illegible]
how he is progressing. Mild excercises may be instituted when muscular spasm and pain have lessened considerably. Heat should be applied locally over the joint fifteen to twenty minutes several times a day. Mild gum-chewing exercises may then be begun, chewing at first a soft and later a harder gum for fifteen to thirty minutes several times a day.



FIG. 130 —Shortness of the lower jaw from juvenile rheumatoid arthritis, producing "bird-like facies"

BIBLIOGRAPHY

1. BALBONI, V. G, HOLLANDER, J L and KYDD, D M: Am. Jour Med. Sci, 212 153, 1946.
2. BAUMGARTNER, W.· Schweiz med. Wchnschr, 76, 809, 1946
3. BAYLES, T. B Med. Clin. N. A, 32, 1357, 1948
4. BAYLES, T B, and RUSSELL, L. A. Jour. Am Med Assn, 116, 2842, 1941
5. BELL, B T. Med Clin N A, 24, 1735, 1940.
6. BENNETT, G. E, COLBY, M C and KENDALL, H. L.· Jour. Am Med Assn, 109, 1120, 1937.
7. CAPONER, N· Brit Med. Jour, 2, 761, 1946
8. COSS, J. A, and BOOTS, R. H. Jour Pediat, 29, 143, 1946
9. DESMARAIS, M. H. L.: Brit. Jour. Phy Med, 10, 116, 1947.
10. ENSLEN, T B · South African Med Jour, 23, 671, 1946.
11. FOGED, J.· Lancet, 2, 1209, 1949
12. GOLDTHWAIT, J. E. Jour Bone and Joint Surg, 4, 469, 1922
13. GRAUBARK, D J., and PETERSON, M. C: Jour. Am Med Assn, 141, 756, 1949
14. GREEN, W. T.· Am. Jour Surg, 44, 201, 1939.
15. HENCH, P. S, and MEYERDING, H. W.: Med Clin N A, 18, 549, 1939.

operation is most likely to succeed if performed when the ...

lages are always degenerated and should be *removed*. *Bleeding during* operation is controlled by the use of the electrocautery.

Instillation of 25 mg. of hydrocortisone acetate suspension intra-articularly at the time of closure of the joint after synovectomy has been suggested by Hollander[35] to minimize the post-operative reaction in the articular tissues.

Selection of Cases for Synovectomy

- This is used most often in cases of rheumatoid arthritis with little or no involvement of the cartilage or bone (with most of the process localized to the synovial membrane)
- Synovectomy at the knee is indicated when synovial tissue and pannus have filled the anterior portion of the knee about the patella.
- Patients with persistent swelling and chronic hydrops which interfere with function are often helped by synovectomy.
- There should be a preceding trial of conservative therapy, including splinting, corrective and muscle setting exercises and *x*-ray therapy, before surgery is performed.
- Do not operate until acute inflammation has subsided
- *Patients will require prolonged postoperative physical therapy.*

Following synovectomy, large compression dressings[63] *consisting* of firm elastic cotton bandages over sterile sheet cotton are used for two days and active motion of the joint is begun several days postoperatively, with weight bearing two or three weeks postoperatively. If there is much pain post-operatively a cast may be applied for four to seven days. If there has been a preceding flexion deformity, a long leg plaster cast is applied in maximum extension after the synovectomy has been performed Early and persistent exercises must be carried out to prevent the development of painful adhesions. If the muscles are weak, walking is carried out at first with plaster cylinders or calipers New synovial tissue forms slowly from the surrounding connective tissue after the synovial membrane has been excised.

Operations for the Relief of Pain—Many surgical procedures have been attempted and are now being tried to relieve pain in severe arthritis The simplest of these are injections of anesthetic solutions into and about joints These give only temporary relief. Anesthetics of longer duration have been only slightly more helpful. Injections into the nerves about the joints have also given only temporary relief. Injections of procaine into and about the sympathetic ganglia have led to *prolonged warmth and improved* blood flow in the arm or leg and in this way have made the patient more comfortable Such injections are indicated particularly in the presence of trophic disturbances and when the extremity is cold constantly.

Osteotomy is used in rare instances in the lumbar spine to correct ankylosis in a position of excessive forward bowing of the spine.[89] The lumbar spine is then hyperextended and a fusion is performed. This, through increased

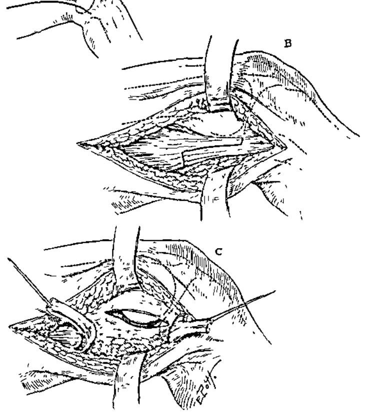


Fig. 134 —Posterior capsuloplasty for fixed flexion contracture of the knee

arthroplasty which may leave the joint unstable and painful The osteotomy is usually performed just below the greater trochanter. Flexion and adduction are corrected and the knee and foot face directly forward. A long plaster spica gi...

ened to the femoral shaft

een maintained.[42]

As an alternative procedure, Buck's extension may be used applying

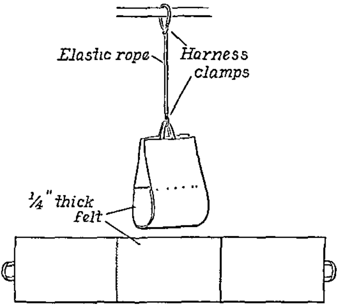


FIG 141 —*Canvas cuff sling suspension* for support of the leg and for assistance in exercises in patients with arthritis of the knees

Traction must be supervised carefully. It must be kept on constantly usually for several weeks to secure correction When the deformity has been overcome, the position may be maintained by a light plaster cast In some patients, the flexion deformity is not completely overcome by serial plaster splints In these, the remaining flexion contracture can often be corrected by gentle manipulation under anesthesia, followed by application of a light plaster cast to maintain this[56]

Caliper Splints.—These are useful in the individual with slight flexion of the knees and in whom one wishes to avoid further flexion when the patient is up and about The main features of these splints are as follows

Lateral steel uprights, extending upward from the heel to the upper thigh on each side of the leg A light combination steel and leather

circulatory embarassment if the knee were fully extended immediately. In such instances, gradual extension of the knee is carried out over a two week period following the operation by several changes of plaster casts, or later manipulation Motion is begun in one week and walking with a support is permitted in two to three weeks. If there is only a narrow range of motion, but the articular surfaces are not greatly distorted, an osteotomy just above the knee with straightening of the knee at the osteotomy site may give a more useful extremity. A posterior capsuloplasty gives the best result if the chief deformity is contracture of the soft parts and there is little destruction or distortion of the articular surfaces. If the articular surfaces are markedly eroded or destroyed an arthroplasty or an arthrodesis are the most satisfactory procedures to consider.

Osteotomy.—Osteotomy (the cutting of a bone) is employed in chronic arthritis chiefly after a joint becomes ankylosed in a faulty position for function In the upper extremity it is used at the finger and wrist joints When the fingers are stiff or subluxated in a manner which prevents closing

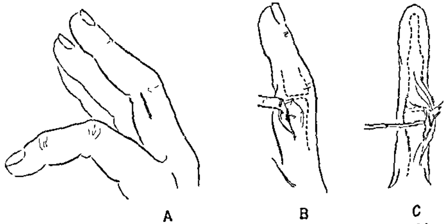


FIG 133.—Release of the capsule in resistant flexion contracture of the finger. (Kuhns, Jour. Bone and Joint Surg 1935)

... the joint line and the fresh-

joint but is always unstable.[61] The newer plastic endoprosthesis may prove

...ends

... has

been prolonged subluxation with ulnar deviation the proximal end of the proximal phalanx must be trimmed also. No interposition material is required at this joint. A useful, painless function is usually obtained.

If the wrist is fixed in palmar flexion and cannot be brought into dorsiflexion by manipulation, a wedge osteotomy through the wrist joint may be necessary The wrist is then fixed for six weeks in 35 degrees of dorsiflexion.[72]

obtain relief from conservative treatment and who are not good risks for major surgery.

At the knee and elbow joints in osteoarthritis, removal of large spurs, loose bodies, thickened synovial tissue or degenerated cartilages (at the knee) is indicated if these cause a serious disturbance in function.

Drilling of the femoral head and other procedures to increase the local circulation have been disappointing. Excision of the patella has seriously disturbed the mechanics of the knee and leads to gradual weakness of the extensor muscles. If the patella is greatly deformed it is made smooth on the under surface and covered with nylon membrane. This gives a much more useful knee than after complete removal of the patella.

THE CORRECTION OF DEFORMITIES IN SPECIFIC JOINTS

The natural tendency for individuals with arthritis of the knee is to flex the knee to decrease pain and to relieve the tension and the pull of the hamstring muscles upon the posterior portion of the joint capsule. This results in shortening of the hamstring muscles and contraction of the capsule, and leads to further flexion of the knee. This deformity usually is begun by allowing a pillow to be placed beneath the knee. Further contractions lead to subluxation of the knee (downward dropping of the tibia on the femoral condyles)and outward rotation of the tibia on the femur.

A flexed knee causes shortening in the extremity, resulting in equinus of the foot and unstable weight bearing. The latter produces abduction and external rotation of the hip to obtain a broader base on which to stand. Later, there follows a tilt of the pelvis and shoulders to the shorter side with lateral deviation of the spine (scoliosis). Thus, flexion deformities of the knee may precipitate a traumatic flat foot, strain of the internal lateral ligament of the knee, knock-knee and traumatic synovitis, later followed by changes in the hip and scoliosis [67]

Canvas Cuff Sling Suspension.—The use of the canvas cuff sling suspension may help prevent subluxation at the knee and is a good method of

down upon its elastic cord, and the leg is slipped through this, with the upper portion of the tibia resting upon the felt padding inside the canvas cuff. This apparatus can be made easily at home. It also serves as a useful

deformities

the patient

to adhere to treatment), conservative orthopedic management may be sufficient to restore these to a practically normal position.

In brief, the technique of such an operation is to divide the bones at the line of the former joint, to smooth and to trim the ends to simulate normal

plastics have proved most satisfactory. Thin nylon membrane has so far been the best material at the knee.[65]



FIG 135 —Temporary arthrodesis of the hip A long triflanged nail is driven through the acetabulum into the ilium

The most favorable age group is from twenty to forty-five· operation under the age of eighteen endangers epiphyseal growth. Arthritis should be inactive for at least six months before attempting operation

In ankylosis of both knees, arthroplasty [8] of one of these may be sufficient, but we have seen many satisfactory results after arthroplasties on both knees When both hips are ankylosed, bilateral arthroplasty may be necessary to permit sitting If both hips and both knees are ankylosed, arthroplasty is advised in both hips before the arthroplasty of the knee.

recommended as motion is then impossible, most orthopedists are opposed to fusion operations on the knees since these cause increased strain on the joints above and below the knee The orthopedic surgeon should aim to preserve or increase joint motion when possible in arthritis rather than to eliminate it.[44]

Supports for the Knee —Elastic bandages —Strapping should be begun below the knee joint and continued upward, covering an area of about 5 inches on each side of the joint A 4 inch ACE elastic cotton bandage is often used in milder inflammation and weakness.

Correction of Deformity at the Knee

1 The knee joint, the largest joint in the body, is often attacked because it is a weight-bearing joint and is frequently subjected to sprain and other injuries

2 Pain, swelling and fixed flexion are the first signs of impending deformity, followed soon by atrophy of muscles and ligaments about the joint, narrowing of the joint space, external rotation, subluxation of the tibia on the femur, fixation of the patella against the femoral condyles, contracture of the hamstring muscles, joint capsule and periarticular structures, and occasionally ankylosis

3 Splint early in a bivalved cast as near full extension as possible Begin exercises as soon as this can be done without pain Apply heat several times a day

4 If flexion deformities are present, these may be corrected by the

valved within forty-eight hours in most instances, using the posterior half as a resting shell The knee joint should be moved through as full a range of painless motion as possible each day

5 If serial casts or plaster shells are used to reduce flexion contractures, these are renewed as improvement occurs

6 When correction of deformity has been obtained, this may be maintained by exercises to strengthen the extensor muscles and by wearing a plaster cast at night The cast and exercises may be discontinued when the muscles are strong and all inflammation has subsided

7 If casts and manipulation fail to correct the deformity, surgery must be considered This may require lengthening of contracted soft tissues If there is bony ankylosis, an osteotomy or arthroplasty may be necessary [12]

anterior and posterior half This is held together in walking by straps or a bandage

10 The atrophied muscles should receive daily physical and occupational therapy After operations, exercise in a Hubbard tank is helpful Later resistive exercises should be given

Osteotomy just above the femoral condyles with extension of the leg at this point is sometimes employed (described earlier) in flexion deformity of the knee. At the foot a wedge osteotomy may be necessary when the tarsal joints are ankylosed and the foot is fixed in valgus and equinus After operation the foot is held in a good weight bearing position for six to eight weeks. A foot support must usually then be worn for some months longer. Exercises and physiotherapy are given after the osteotomy has healed, to obtain muscular strength and good function.

Articular Resections.—Resection of the proximal or distal portions or both sides of a joint is employed in the treatment of chronic arthritis It is particularly of value in the smaller joints of the hand or foot. When these joints are grossly distorted undue pressure comes on a portion of the joint and causes pain. Articular resection is used most often in the foot particularly at the metatarso-phalangeal joints, when the forefoot is spread and a great amount of pressure comes upon the under surface of the metatarsal heads Usually only the 2d, 3d and 4th metatarsal heads are removed but in exceptional cases all may be removed

In the hand with ulnar deviation and subluxation of the proximal phalanx medially, the metacarpo-phalangeals are resected. Here the distal end of the metacarpal bone is trimmed squarely across as well as the proximal end of the proximal phalanx. This permits the finger to move in its normal axis. With severe painful arthritis of the acromio-clavicular joint at times it is necessary to resect 1 inch from the distal end of the clavicle. When pronation and supination are prevented by ankylosis of the proximal or distal radioulnar joints or both of these, the distal end of the ulna or the radial head is resected This usually permits adequate supination. Articular resections are not performed in the major joints of the body since they lead to instability in these larger joints.

Arthrodesis —Arthrodesis[1,4]—the operative fusion of a joint in the best position for use—is performed only occasionally in chronic arthritis Arthrodesis after osteotomy has already been mentioned in relation to the finger and wrist joints. As a temporary measure in hopelessly damaged, painful hips in rheumatoid spondylitis the hip can be made comfortable by driving a long tri-flanged nail through the femoral neck deeply into the acetabulum. Arthrodesis has at times been advocated in severe osteoarthritis of the hip. In our experience it has brought on severe osteoarthritis in the low back or in the hip joint on the other side It has relieved pain only for a short time. Arthrodesis of the knee often leads to increasing arthritis above the knee on the same side and to sprain in the joints of the other side[83] About the foot, arthrodesis of the ankle and posterior tarsal joints in the best position for weight-bearing are useful procedures and often relieve pain and most of the disability in these joints However, in severe arthritis these joints tend to fuse spontaneously. The surgeons task is to keep the foot in a good position for walking until ankylosis takes place in these joints If firm ankylosis fails to occur arthrodesis should be done

Arthroplasty.—This is an operation to form a new, moveable joint when ankylosis is present or the articular surfaces are greatly distorted. Satisfactory results have been obtained in ankylosis of the hips, elbows and knees; results have been less good in the fingers and jaw.

thigh band attached to the upper end of the uprights. Leather straps *anteriorly at the knee joint.* Leather strap at the ankle joint to *maintain the lateral* uprights in place. Sockets in the lateral aspects of the heels (anteriorly) into which the lower portions of the calipers fit.

Calipers can be used to correct slight flexion deformity (less than 30 degrees) either in bed or while the patient is ambulatory. They are used for several periods a day with the knee held in as near to full extension as possible.[61]



Fig 142 —Caliper splint to correct flexion at the knee Also to maintain full extension in walking when the muscles are weak

Recent [illegible] ed to [illegible] ascial [illegible] hods of correction have become available.[7]

If deformities do not respond to splints, traction, or manipulation in a reasonable period of time (three to six weeks), one may resort to surgical procedures including capsuloplasty, tendon lengthening, and in some cases osteotomies and arthroplasties, if the disease is inactive and the general condition of the patient permits[53]

Release of the posterior side of the joint capsule produces good results when the posterior capsule is tight from adhesions, and flexed knees have motion with the patella free. Synovectomy is successful in some selected cases where pannus has formed in excess between the tibia and femur and synovial tissue has filled the suprapatellar pouch around the patella. Arthroplasty may be required if there is ankylosis. Arthrodesis is seldom

much less when plastic or metallic molds are fitted over the articular surfaces. Vitallium (65 per cent cobalt, 30 per cent chromium and 5 per cent molybdenum) is one of the best metals to employ in bone surgery It results in no irritation of the surrounding soft tissue or bone and no discoloration or staining of the structures.

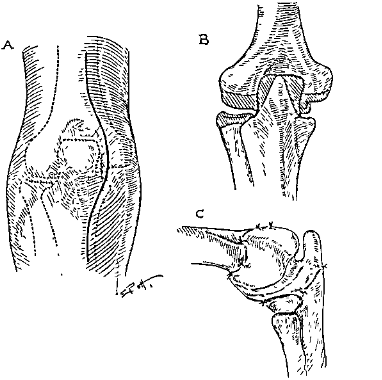


FIG 137 —Sketch of arthroplasty of elbow with interposition of fascia

When the femoral head is greatly misshapen either from disease or injury during the growing years, or from extensive, bony overgrowth in osteoarthritis, it is always difficult and sometimes impossible to reshape the femoral head in a satisfactory manner In such instances a more nearly

satisfactory The femoral neck is sawed so that its cross surface projects in a valgus position Any anteversion or retroversion of the neck is also corrected The plastic appliance is driven on to the femoral neck to obtain as nearly a normal alignment as possible. This procedure has given painless hips with almost normal function It has proven to be of particular value in severe osteoarthritis of the hip.

the hip due to a flexion contracture Muscular spasm results in flexion, adduction and in some cases external rotation.[1]

A fixed flexion and adduction deformity of the hip produces a functional

Measures of value in arthritis involving the hip joint include.

Correction of Deformity at the Hip

1 The first symptom may be pain referred to the anterior portion of the thigh and mesial aspect of the knee
2 Examination usually reveals muscle spasm and a flexion deformity If improperly treated, the process will usually progress to permanent flexion deformity and marked muscular atrophy with adduction and external rotation
3 Hip flexion may be corrected in some early cases by having the patient lie prone (on his abdomen) in bed at least thirty minutes several times per day

overhead frame This permits the patient to exercise that leg almost as easily as in a Hubbard tank
6 In acutely painful hip joints, it may be necessary to apply a unilateral or bilateral plaster spica to secure relief It may be changed every week or ten days as an improved position is secured
7. If a deformity is present without bony ankylosis, much of this can often be corrected by gentle manipulation under anesthesia with maintenance of the correction by plaster spica casts
8 When the patient becomes ambulatory, a light-weight spica cut into an anterior and posterior half may be worn, this may be removed when desired for physical therapy Crutches should be used at first

1 Elimination of weight bearing by rest in bed; this relieves muscular spasm and pain
2 The patient should lie on his back on a firm, non-sagging mattress most of the time. Lying flat on the face also stretches a flexion deformity at the hip.[39]
3. Maintain extension of the hip by sand bags, casts or traction [47]
4. A long plaster spica is the most effective early method of correcting flexion adduction and external rotation at the hip. The spica can be changed when further correction of the deformity is possible

A layer of 4 inch elastic bandage is wound loosely around the knee (for 4

Elastic knee supports, with or without lacing, are often of great value in weak knees; if lacing is used, the felt pads as described above may be used on either side of the patella.[41]

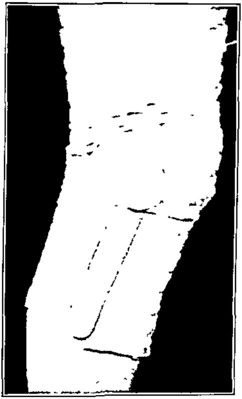


FIG. 143 —Elastic knee brace, with lacing and side hinges used for ambulatory support in arthritis of the knee.

Elastic knee supports with hinged side joints: In place of the lateral felt pads, thin steel strips may be incorporated into the outside of the laced

steel;

The

supports may be laced up the front.[7]

Correction of Deformity at the Hip.—In arthritis of the hip, pain may be referred to the knee or to the thigh medially. The first sign of deformity in arthritis of the hip is inability to extend the leg completely at

Nylon membrane has been used because fascia will not last but disappears rapidly under weight bearing. After reshaping the articular surfaces a single layer of nylon is attached over the articular surface and condyles of the femur. It has caused no irritation or reaction in the knee In ankylosis, destruction of the articular surfaces or in severe osteoarthritis it has frequently resulted in a painless knee joint with a wide range of motion These arthroplasties of the knee have been observed for as long as eight years with no deterioration in function [55]

CONSERVATIVE ORTHOPEDIC TREATMENT OF OSTEOARTHRITIS

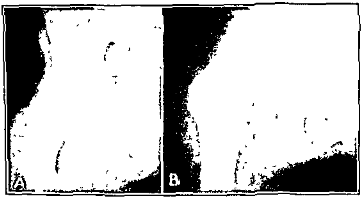


FIG 139 —*A* Fusion of the hip joint *B*. The same hip after vitallium mold

In osteoarthritis, the orthopedic surgeon must deal with stiffness and pain on motion more than with deformity Special measures are often required for osteoarthritis of the spine,[47] hip and knee—less commonly of the foot, elbow and wrist.

In the spine, heat is the most effective measure in decreasing stiffness Any other measures which increase the peripheral circulation are of value Support to the back which prevents the minor sprains so common in osteoarthritis,[53] often keeps these individuals in active work When there is marked stiffness and much pain complete rest on a firm bed for a few days to a week usually relieves symptoms Occasionally, a plaster shell, rarely a plaster jacket, is required When symptoms persist or recur frequently a well-fitted spinal brace with shoulder straps or a firm back-laced corset will often prevent a recurrence of symptoms Exercises are given as soon as soreness subsides to improve the posture, to correct the general alignment of the spine and to strengthen the spinal musculature Osteoarthritis of the cervical spine often produces severe neurological symptoms over the

through the tuberosity down the steel uprights. The lateral steel rods may end below as in the caliper splints (in the heel of the shoe) or in a rubber covered base which acts as the lower end of the apparatus, and upon which weight is borne. In milder deformities a corset belt or a leather pelvic belt can be used. In elderly individuals to relieve pain a short leather spica can be used.



FIG 145.—Support for lumbo-sacral spine. Anterior-posterior pad, with leather reinforced posterior support, and anterior abdominal pad, used for arthritis of lumbar spine or pelvic joints.

Correction of Deformity at the Foot.—Strain or deformity may result from deformity of the knee or hip. Flattening of the so-called anterior arch frequently results from flexion of the knee. The common deformities at the foot are valgus, spread of the forefoot, equinus, deformities of the toes and ankylosis.[6]

The feet are pulled into plantar flexion (equinus) in patients confined to bed because of the weight of tight bed clothes, gravity and the pull of the strong gastrocnemius muscle.

Support for the Low Back.—**(Lumbosacral and Sacro-iliac Joints).**—Adhesive strapping is the simplest form of support for the low back. A proper corset may be required. This should be a back lace corset which tends to

In the upper extremity arthroplasty is performed chiefly at the elbow.[?] At the finger[85] and wrist joints moveable joints can be obtained but these are usually so unstable that they interfere with use. Fusion of the wrist or interphalangeal joints at about 30° of flexion leads to better function than arthroplasty. At the elbow the bones are reached by a posterior incision

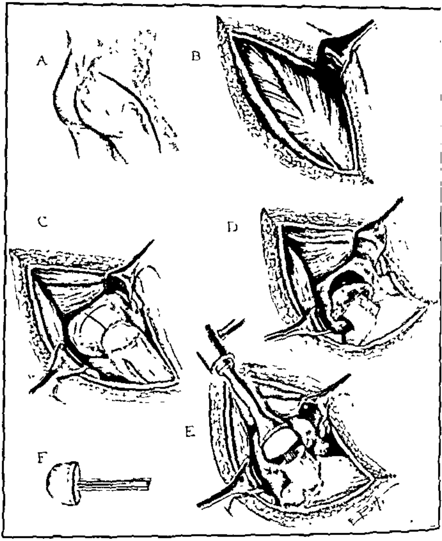


Fig 138 —Replacement of femoral head with acrylic or metal prosthesis *A* Incision *B* Exposure of extensor muscles *C*, Resection of muscles and articular capsule *D*, Sectioning of deformed femoral head *E* Prosthesis being attached to femoral neck *F* Prosthesis to replace femoral head

After the ankylosis has been chiseled away, the bone ends are reshaped and a single layer of fascia is fitted over the exposed bone

At the knee, fascia wears out quickly with use. Nylon membrane 6/1000 of an inch thick has withstood wear satisfactorily and is not irritating to the tissues.

5. Occasionally, traction with Buck's extension may be used early, or in the presence of severe pain, with the leg in adduction.
6. Apply hot fomentations for thirty minutes, 2 or 3 times a day.
7. When complete extension of the hip has been obtained, continue the use of a bivalved spica and remove this daily for exercises and physical therapy.[80]
8. The patient should not get out of bed until he can keep the thigh fully extended (i.e., flex one knee with the other leg in full extension without arching the back).
9. Weight bearing is not permitted until all muscular spasm has disappeared, and partial weight bearing is permitted with use of crutches
10. If necessary, use surgical procedures such as manipulation under anesthesia (followed by use of a plaster cast enclosing the entire affected leg and pelvis); or operative procedures.[12]

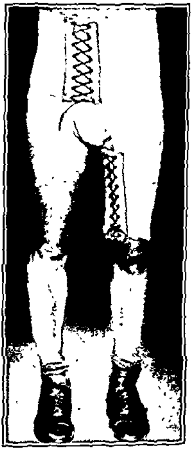


FIG 144.—Unilateral plaster hip spica to provide support in arthritis of the hip joint Lacing permits easy removal for bathing or physical therapy.

Support for the Hip.—This may be obtained by a figure-of-8 application of wide elastic bandage to the pelvis and thigh as a spica, with strips of ½ inch thick felt incorporated in the bandage at the lateral side.

physiotherapy, exercises, and the use of support about the joints—usually gives comfort. When these measures fail surgery should be considered if the general condition of the patient is good.

Surgery is performed in osteoarthritis most commonly at the hip and knee. In younger individuals, particularly in those with osteoarthritis only in one hip, an arthroplasty with the removal of osteophytes about the acetabulum and the fitting of a metallic cup over the femoral head or the replacement of the femoral head with a plastic prosthesis frequently relieves pain and is followed by good function [34]. In older individuals, considerable relief of symptoms follows an osteotomy just below the femoral neck. The femoral shaft is displaced inward. The leg is brought into about 20 degrees of abduction and the external rotation is corrected. Usually a stainless steel blade plate holds the fragments in the improved position and no plaster spica is required. Non-weight-bearing exercises are started in two weeks. Weight bearing with crutches is permitted in four weeks [31].



FIG 140 —Severe osteoarthritis of the hip joint with osteotomy below the femoral neck

is no appreciable disturbance in function despite the weakening of the adductors. Adductor spasm increases deform

Correction of Deformities of the Spine

1. In *degenerative joint disease* in middle-aged or older patients, *most patients respond well to* heat, rest, support (with adhesive strapping, corsets, steel back braces if required with elastic shoulder straps if the thoracic spine is involved) and correction of poor posture (if the latter is present)
2. In *rheumatoid spondylitis*, the sacro-iliac joints are usually affected early and may fuse before marked involvement of the spinal column is noted Chest motion may be limited and vital capacity be decreased by involvement of the rib articulations, the patient may be unable to turn to either side without moving the whole body
3. **Roentgen therapy is one of the most useful measures in relieving pain and muscle spasm and in stopping the progress of a rheumatoid spondylitis**
4. In rheumatoid spondylitis, the patient should use a *firm, non-sagging bed* and may obtain relief by the use of *hot fomentations* to the back. Boards may be placed between a thin, hair mattress and the springs In bed-ridden patients, a *posterior plaster body shell* may suffice temporarily
5. The patient with very severe rheumatoid spondylitis may require immobilization in a plaster body jacket, and later in a leather or plastic body jacket The jacket may be removed for the use of heat and massage. Plaster jackets are changed as further correction occurs
6. In *arthritis of the cervical spine*, rotation and lateral tilting of the head may be prevented and corrected by a *Thomas collar*, felt collar or plaster head piece If the patient with cervical arthritis is confined to bed, sand bags may be used to hold the head in place and *head traction* may be required if severe spasm has occurred or if severe pain is present *Hot fomentations* are useful in relieving pain and muscle spasm
7. The special positions mentioned under Posture will help to correct deformities
8. Postural and breathing exercises should be continued until soreness is gone
9. Operative correction of spinal deformity is required in seriously deformed patients

5. The second strip begins on the other side and runs downward in the opposite direction
6. At least six strips are used on each side, alternately applying the right and the left, each one overlapping the previous strip on that side. Each succeeding strip begins further laterally
7. This will remain in position better if finally two or three strips of adhesive are placed longitudinally over this on each side of the spinal

irritation. Not infrequently there is bony proliferation about the neural foramena with exostoses projecting vertically producing symptoms indistinguishable from ruptured intervertebral disc. Collapse of the intervertebral discs is a common finding in severe osteoarthritis of the spine, and with this some herniation of the discs may occur. Laminectomy may be necessary in such instances.

In the hip, osteoarthritis leads to pain, stiffness and increasing adduction and external rotation. *In the milder cases, rest, avoidance of weight bearing,* or the use of crutches may relieve symptoms. Reduction of weight,[51] use of a tight corset, and correction of postural faults are also helpful. Traction to the legs, heat and exercises are valuable adjuncts[53] More advanced cases often need the aid of orthopedic measures described below.

At the knee, the joint which most frequently produces serious symptoms in osteoarthritis, the intra-articular injection of Hydrocortisone[54a] has proven of palliative value, but should be used in conjunction with rest, support from elastic bandaging, and correction of static deformities of the feet In more severe cases these measures will not be adequate, and operative interference will be necessary

When symptoms persist at the knee joint after complete rest, particularly limitation of full extension, swelling and muscular spasm, some form of impingement on the articular surfaces should be suspected In most *instances a pinch of hypertrophied synovial membrane* will be the cause.

vectomy, removal of loose bodies and extirpation of *displaced semilunar* cartilages often relieve symptoms and lead to subsequently improved ... destroyed better

In other joints, support is usually the most effective treatment. Hope...

In the toes,

dorsiflexion

fort. **In the fingers,** heat and the avoidance of rapid or prolonged activity keep symp*toms from becoming severe.* Firm adhesive strapping will relieve symptoms temporarily both in the finger joints and at the wrist. The elbow can be kept comfortable with a firm bandage or strapping. When symptoms are persistent a hinged elbow cast should be worn. With severe limitation *from bony overgrowth (osteophyte formation),* an arthroplasty of the elbow gives a painless joint with excellent function.

RECONSTRUCTIVE OPERATIONS IN OSTEOARTHRITIS

Surgery is employed much less frequently in osteoarthritis than in rheumatoid arthritis. The patients are usually old and present many other degenerative processes besides the arthritis Conservative therapy—rest,

hold the low back flat and the lower abdomen in. Low back and abdominal *supports and corsets are valuable only when they are fitted properly* to the individual patient in an anatomic and physiologic manner.[2] A firm canvas belt may be worn. This should come firmly over the sacro-iliac joints. For more severe disability a back brace may be required. This should hold the low back relatively flat and the front pad should hold the lower abdomen in. The physician *should determine that the spinal support is properly fitted* and worn, otherwise it is of little value. With any disability in the low back, a firm bed should be used. Postural exercises should also be prescribed.[15,35]

Correction of Deformities of the Feet

1. [illegible]
2. [illegible] obtained
3. Use heat, or warm foot baths to lessen inflammation.
4. Give exercises to stretch contractures and strengthen muscles.
5. Strapping of the foot with adhesive plaster which is changed at weekly intervals will often correct spasmodic valgus
6. When deformities are resistant, manipulation of the foot should be performed After manipulation the foot is held in the corrected position with a plaster cast for one week. This is followed by exercises as soon as soreness subsides.
7. Ankylosis in a faulty position for weight bearing can be corrected by wedge-osteotomy
8. Operations are used to correct hallux valgus, hammer toe and dislocation of the metatarsal heads After operation or other correction of deformity, support is given to the foot until normal strength returns
9. Crutches are often required at first when walking is resumed
10. Proper shoes are described in the chapter on Painful Feet

Support for the Thoracic Spine.—Adhesive strapping is the simplest form of support If necessary, one may use a back brace with shoulder straps By the use of plaster jackets, spasm of the erector spinae muscles is [illegible] If the pa- [illegible] is used, this [illegible] ward bowing [illegible] looser above [illegible] It may be [illegible] in

Strapping for the Thoracic Spine.—1. The part to be strapped is shaved if necessary and is then painted with compound tincture of benzoin.
2. Adhesive tape 2 inches wide is used.
3. The strapping runs obliquely from the clavicle downward across the back to the loin.
4. The first strip begins at the base of the neck and runs downward and inward.

adhesions about the capsule of the shoulder joint. This is discussed elsewhere (See chapter on The Painful Shoulder, p 944) If proper positions

under anesthesia or vigorous exercises to stretch the contracted muscles This must always be followed by postural exercises and exercises for the shoulder to prevent a recurrence

It is best to keep the shoulders abducted and externally rotated to lessen

No pillow or only a small pillow is allowed under the head

Correct Deformities at the Shoulder by

1. Use of a flat, firm bed, with only one small pillow, if any, beneath the head
2. The patient should lie with his *hands clasped beneath his head* several times a day for short periods such a position permits abduction and external rotation of the shoulder
3. In arthritis involving the shoulder, *support the arms* by sand bags, pillows or plaster splints *in as close to full abduction* and *external rotation* as possible to prevent forward drooping of the shoulder with consequent adduction and internal rotation of the arm If the patient is ambulatory and other joints are not involved an airplane splint may be used More abduction is obtained by changing the splint
4. Pain and muscular spasm are relieved in the active stage of an arthritis

sides

skilled hands) to obtain increased range of shoulder motion if conservative treatment fails Shoulder motions must be continued at least several times a day following manipulation to prevent recurrence of adhesions Procaine hydrochloride 1 per cent solution may be *injected into the joint and periarticular structures to minimize pain* and allow early exercises

7. Ankylosis is rare at the shoulder joint, surgical intervention is rarely necessary for restoration of motion.

washes out more than proportional amounts of carbon dioxide Other changes in the acid-base balance induce a mild alkalosis Oxygen consumption is increased, and fluid transfer across the capillary wall is accelerated [103] Absorption of exudates is brought about by the latter mechanism. As with local heating muscular relaxation occurs A sedative effect on the peripheral and central nervous systems has been noted The most obvious result of the application (to the layman) is sweating, and this has given rise to the popular idea of "elimination" as a means of therapeusis

Value of Heat in the Treatment of Arthritis.—A proper amount of heat increases blood and lymph flow in the affected area, which aids the removal of waste products and hastens the absorption of inflammatory exudates Improvement in local nutrition is brought about. Tissue relaxation, especially that of the muscles, which may be in spasm, affords comfort, and even some mild sedation An analgesic effect is almost always produced Available forms of local heat include heat lamps (luminous), infrared lamps (non-luminous), hot wet compresses or poultices, hot water bottles, electric heating pads, hot bags of salt or sand Luminous heat penetrates somewhat more than infrared,[67] that from a tungsten filament bulb to 3 to 5 mms or approximately to the capillary bed It is more effective in stimulating reflex vasodilatation than the non-luminous infrared generators.[67] Application of heat to the extremities of a diabetic, or to any patient with loss of sensation, scars, or impaired circulation entails a risk.

A. Forms of Applying Dry Heat

Heat Lamp.—The simplest type consists of a cup-shaped polished reflector and a clamp (or handle) which may be attached to a chair back or bed A 250-watt Mazda CX bulb is connected with an ordinary elec-

"hot spots" are present in such lamps where heat energy is more intense Burns are more apt to occur than with lamps providing a more even distribution of heat. Similar lamps attached to an adjustable stand or with a goose neck afford more flexibility and permit perpendicular application to

or tungsten filament provide a uniform heat, distributed evenly over all areas beneath the reflecting surface. An inexpensive model for home use

60-watt bulbs. These receptacles are connected in multiple with heavy

A Thomas collar may prove very useful in the patient with arthritis involving the cervical spine.[23] If felt is not available, a piece of cardboard

bone and clavicles. The collar may be fastened in the back of the neck by a gauze tie or buckles. This will relieve some of the strain on the cervical spine by allowing the weight of the head to be borne in part directly upon the shoulders. If pain is severe or there is much spasm a plaster head piece may be made and worn constantly.[37] In patients with severe spasm in the cervical muscles, it may be necessary to employ traction by means of the Sayre head sling attached by a cord, passing over a pulley at the head of the



FIG. 146.—Leather Thomas collar used for ambulatory support in arthritis of the cervical spine.

bed. Weights of about 5 pounds are used to produce traction as tolerated. When muscular spasm has been relieved by traction, a light felt or leather collar may be utilized. Exercises and physiotherapy are prescribed.[14]

Support for the Entire Spine.—One may utilize plaster casts or shells, plaster or leather jackets or a Bradford frame. A firm non-sag mattress and fracture boards should be used whenever there is back disability.

Correction of Deformities at the Shoulder.—Inflammations around the shoulder joint lead to adduction and internal rotation of the arm.[64] Stiffness of the shoulder may be caused by adhesions around the bursae and joint and contractures of surrounding tissues (capsule, muscles, bursae). There is atrophy of the deltoid muscle. True ankylosis of the shoulder is rare in arthritis.

Limitation of motion at the shoulder usually results from calcification in the supraspinatus tendon with an associated subdeltoid bursitis, or from

Among the effects to be noted by local application of heat are dilatation of the blood vessels, hyperemia, and local sweating. Heat relaxes muscles, improves local nutrition, and is frequently analgesic. It may cause harm if

acutely inflamed joint when the overlying skin is hot or red.[14] The incorporation of a medicinal agent, e.g., hot saturated solution of magnesium sulphate, often greatly enhances the effect of local heating. It may be kept hot by repeated application or the use of a radiant lamp or hot water bottle.

Application of Heat in Rheumatoid Arthritis

In the acute stage, with red, hot, swollen, tender joints, if the patient can be made comfortable by the use of splints, rest and medication, do not apply heat to the joints. If, in spite of such care, the joints remain painful and there is no tendency toward quiescence of the disease, one may apply hot wet dressings of a saturated solution of magnesium sulfate to the affected joints for periods of thirty minutes several times each day. If hot fomentations result in further pain, they should be discontinued at once. If the patient does not permit the weight of the dressing upon the affected joints (because of pain) employ radiant heat as infrared rays for from five to twenty minutes several times a day, unless this produces an aggravation of symptoms.

In the acute stage, *do not employ* diathermy or hot baths. Poor results have been obtained to date with fever therapy in rheumatoid arthritis.

As the patient improves and the disease passes *into the subacute or chronic*

the disease is more or less generalized, use the hot water bath (gradu-

within twenty-four hours by an exacerbation of symptoms, this should be modified or discontinued.[32]

For involvement of individual joints in chronic or subacute rheumatoid arthritis, some of the common heating methods which may be employed include bakers (which can be made inexpensively), ordinary small hand heating lamps, infrared lamps, hot paraffin applications, contrast baths, conventional diathermy and even short wave therapy. For home therapy, use a heat lamp for thirty minutes two or three times a day at a distance of comfortable toleration over the affected joints.

Systemic application of heat is more generally used in arthritis than in most other medical conditions. Its primary effect is on the circulatory system where one of the early developments is a rise in pulse rate and a slight drop in blood pressure. Maximal vasodilatation is not reached before twenty to thirty minutes.[136] Pulmonary ventilation is increased, roughly

physical therapy and exercise. One may employ elastic bandages, elastic elbow cuffs or elbow braces to relieve pain and muscular spasm

The common deformities at the elbow are flexion with pronation of the forearm Limitation of extension is common but ankylosis is seen only after very severe arthritis

a useful joint.[66]

Arthritis Involving the Hands
1 Prevent ulnar deviation of the hand and faulty position of the fingers by the use of a *molded plaster splint* extending from the forearm to the finger-tips, with dorsiflexion of the wrist and moderate flexion of the fingers, a flange on the ulnar edge at the hand prevents ulnar deviation of the fingers The palmar arches are molded The splint is made convex at the palm
2 *Warm paraffin dips* may be used in the acute or chronic stage to relieve muscle spasm and pain (See instructions in chapter on Physical Therapy for details)
3 *Finger exercises* daily will aid in maintaining motion These include squeezing a sponge, attempting to extend the fingers, attempts to separate the fingers from each other, and touching the tip of each finger to the tip of the thumb several times per day
4 Wood carving, needle work and other forms of *occupational therapy* are very helpful in these cases, and emphasis should be placed on extension exercises
5 The *"clam-shell" cuff* devised by Swaim to prevent interphalangeal and metacarpophalangeal subluxations consists of a piece of felt and a plaster slab which is molded and permitted to harden around the subluxated joints in corrected positions *When dry, it may be held on the* hand with an elastic band or webbing strap It is easily slipped off and on the hand
6 Avoid excessive weight bearing on the outstretched hand as this may produce a "flat" hand with loss of the palmar arch This can be helped by a wrist strap or a well molded leather wristlet which extends to the metacarpal heads
7 If stiffness of the hand is inevitable, it should be placed in a position of dorsiflexion at the wrist, flexion at the metacarpophalangeal joints, 30° flexion of the fingers and apposition of the thumb

Manipulation of the elbow usually leads to further stiffness if performed in the acute phase of an arthritic process In selected cases it can be used when the disease is quiescent In a few cases the biceps humerus tendon is lengthened. In severely damaged or ankylosed elbow joints an arthroplasty should be performed [23]

Wrist and Hand —The usual deformity at the wrist is palmar flexion [40] The wrist joint may become limited in motion or ankylosed. The carpal bones may fuse with the palm flat (obliteration of the palmar arches) The common deformities at the metacarpophalangeal joints are ulnar deviation and subluxation Flexion and sometimes ankylosis may occur in the fingers

sorption of exudates is brought about by the latter mechanism. As with

popular idea of "elimination" as a means of therapeusis

Value of Heat in the Treatment of Arthritis.—A proper amount of heat increases blood and lymph flow in the affected area, which aids the removal of waste products and hastens the absorption of inflammatory exudates. Improvement in local nutrition is brought about. Tissue relaxation, especially that of the muscles, which may be in spasm, affords comfort, and even some mild sedation. An analgesic effect is almost always produced. Available forms of local heat include heat lamps (luminous), infrared lamps (non-luminous), hot wet compresses or poultices, hot water bottles, electric heating pads, hot bags of salt or sand. Luminous heat penetrates somewhat more than infrared,[87] that from a tungsten filament bulb to 3 to 5 mms. or approximately to the capillary bed. It is more effective in stimulating reflex vasodilatation than the non-luminous infrared generators.[87] Application of heat to the extremities of a diabetic, or to any patient with loss of sensation, scars, or impaired circulation entails a risk.

A. Forms of Applying Dry Heat

Heat Lamp.—The simplest type consists of a cup-shaped polished reflector and a clamp (or handle) which may be attached to a chair back or bed. A 250-watt Mazda CX bulb is connected with an ordinary electrical ou[illegible]

lamp [illegible]

this typ[illegible]

Conseq[illegible]

"hot spots"

areas beneath the reflecting surface. An inexpensive model for home use

sheeting are set two double receptacles in which are placed a total of four 60-watt bulbs. These receptacles are connected in multiple with heavy

tion. Abduction of the shoulder in ambulatory patients can be maintained by a plaster or metal splint which holds the shoulder in 90 degrees abduction. When an arthritis becomes quiescent, deformities may be overcome by exercise, traction or manipulation under anesthesia (See Chapter on the Painful Shoulder).

Management of Arthritis of the Elbow

1. The elbow joint is often misused; deformity and limitation of motion may result from making the elbow a weight-bearing joint when the arthritic patient uses a cane or supports himself by holding on to tables and chairs.
2. In the acute stage, rest the arm and hand in a *molded plaster splint* in extension if possible. Active or guided *motion* must be performed several times a day to prevent formation of dense adhesions.
3. If there is any suggestion that ankylosis may occur, the joint should be placed in a useful position: usually slightly less than a right angle *with supination of the forearm (or a position midway between supination and pronation).*
4. Apply *hot fomentations; gentle manual stretching* may be attempted [illegible] may also be accomplished [illegible] nd [illegible]
5. [illegible] be acute stage of arthritis. It is occasionally of value when the disease is quiescent.
6. [illegible] may be preformed if ankylosis [illegible] the olecranon

Prevention and Treatment of Wrist Deformity

1. Gravity and the pull of the flexor muscles produce a deformity of pal[illegible] the normal grasping power of the hand
2. [illegible] ° of dorsiflexion, [illegible] mar crease, the splint may include the fingers if these are painful and swollen
3. *Remove the splint at least once a day* to permit several active motions
4. If marked pain and muscle spasm are present, *hot fomentations* may be applied several times per day.
5. If deformity is present, a series of cock-up splints may gain increased dorsiflexion
6. If ankylosis is inevitable, allow the wrist to fuse in a useful position and, if necessary, manipulate the wrist under anesthesia to obtain sufficient correction before firm union of the bones occurs
7. If the bones have ankylosed in a bad position, this may be corrected surgically producing a fusion with 30° of dorsiflexion
8. With severe damage or weakness a leather wristlet should be worn

Correction of Deformities at the Elbow.—Contractures may be prevented by the application of a molded plaster splint to the flexor surface with the elbow in full extension; this may be removed several times each day for

Among the effects to be noted by local application ...

applied injudiciously, and may even act as a f...

poration of a medicinal agent, e.g., hot saturated sol...

Application of Heat in Rheumatoid Arthritis
In the acute stage, with red, hot, swollen, tender joints, if the patient can be made comfortable by the use of splints, rest and medication, do not *apply heat to the joints* If, in spite of such care, the joints remain painful and there is no tendency toward quiescence of the disease, one may apply hot wet dressings of a saturated solution of magnesium sulfate to the affected joints for periods of thirty minutes several times each day If hot fomentations result in further pain, they should be discontinued at once. If the patient does not permit the weight of the dressing upon the affected joints (because of pain) employ radiant heat as infrared rays for from five to twenty minutes several times a day, unless this produces an aggravation of symptoms
In the acute stage, *do not employ* diathermy or hot baths. Poor results have been obtained to date with fever therapy in rheumatoid arthritis
As the patient improves and the disease passes *into the subacute or chronic stage* (as judged by sedimentation rate, symptoms, joint swelling and ... thirty minutes, depending upon his tolerance. If the bath is followed within twenty-four hours by an exacerbation of symptoms, this should be modified or discontinued.[32]
For involvement of individual joints in chronic or subacute rheumatoid arthritis, some of the common heat methods which may be employed ... l hand ... ntrast ... For home therapy, use a heat lamp for thirty minutes two or three times a day at a distance of comfortable toleration over the affected joints

Systemic application of heat is more generally used in arthritis than in most other medical conditions. Its primary effect is on the circulatory system where one of the early developments is a rise in pulse rate and a slight drop in blood pressure. Maximal vasodilatation is not reached before twenty to thirty minutes.[186] Pulmonary ventilation is increased, roughly

trained for a sitting occupation Others may be served best by a stiff
others

. For
others, job-retraining, with the advice and aid of state and federal experts in this field, may be required. Occasionally the patient will raise his economic status in spite of terrific handicap, *e.g.*, a laborer, stricken with rheumatoid spondylitis with marked limitation of motion in shoulders and hips, became an expert draftsman [16]

Physiotherapy, apparatus and surgery sometimes fail to accomplish much of their objective In such instances less strenuous occupations must be planned. But usually a fairly close estimate of the outcome of treatment

BIBLIOGRAPHY

1 ALBEE, F H Internat Coll of Surg, 5, 17, 1945
2 BAKER, L D South Med Jour, 36, 180, 1943
3 BANKART, A S B Proc Roy Soc Med, 36, 329, 1943.
4 BASTOW, J
5 BELL, B T
6 BISGARD, J D
7 BERKHEISER, I
8 BROOKE, R Brit Jour Surg, 24,
9 BURT, H A Proc Royal Soc Med, 18, 187, 1950
10 CAMERON, D. M and HENDERSON, M S *Proc Staff Meet, Mayo Clinic*, 15, 561, 1940
11. CAMPBELL, W C Surg, Gynec and Obst, 55, 747, 1932

135, 40, 1947

ngland Jour Med, 224, 150, 1941
20 FISHER, A
21 FLETCHER,
22 GHORMLEY,
23 GILLIS, L
24
25
26
27
28
29
30
31

low.[155, 217] Lamps producing a large amount of near infrared energy are the 250 watt CX Mazda bulb, the 250 watt carbon filament bulb, and the newer 250 watt reflector heat lamp in addition to the 500 to 1,500 watt tungsten filament bulbs. The shape of the reflector is an important item in

between the generator and the patient's skin depends on the tolerance and the sensitivity of the part Infrared lamps are the least effective means of heating the hands, forearms, and feet because loss of heat is rapid from those surfaces of the part not directly exposed One of the forms of hydrotherapy or a simple conductive device may be superior

The above methods of applying dry heat all depend for their effectiveness on the emission of infrared rays They are more economical, easier to manipulate, and less dangerous than long or short wave diathermy Direct inspection of the part being treated is always possible Effectiveness of the treatment may be enhanced or varied by the application of hot wet compresses or counterirritants. The former may be kept continuously wet by the addition of water to replace that lost by evaporation

This form of treatment should usually be given several times daily, but it may be used at low intensity as a continuous application in some conditions. Each period should

burning sensations during treatment indicates that the lamp should be moved farther from the patient.

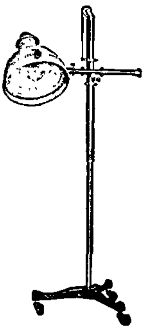


FIG 148 —Large luminous heat generator on stand (Courtesy of The Burdick Corporation)

Electric Cabinet Baths.—These are used only in robust patients Treatment lasts from five to fifteen minutes and is followed by a warm shower and gentle massage or a salt rub. The patient must then lie down and rest for an hour during which time he gradually cools off. Such baths may be employed once or twice weekly Their value in chronic arthritis is doubtful, but may afford some relief to the patient with primary fibrositis

Hot Air Devices.—1 **Hot Air Douche.**—One may use an ordinary hair drier to blow a current of hot air against painful joints (such as fingers and hands) for periods of ten to thirty minutes [129] Penetration of heat in this method is limited.

2. **Heating Jacket** —It is very difficult to heat all surfaces of the shoulder by ordinary means because of its irregular contours In using radiant

The use of a plaster or metal cock-up splint will usually prevent flexion deformity at the wrist. The wrist is held in about 30 degrees of dorsiflexion.

t

t

slight flexion. A flange is placed on the ulnar side to present ulnar deviation of the fingers. The distal and proximal transverse arches and the longitudinal palmar arch are molded. This is done by making the palmar area of the splint dome-shaped.

may be used during

moved at intervals f

splint can be made e

will harden within five to eight minutes. This splint will prevent the common tendency toward the ulnar deviation of the fingers.

A small cuff of plaster may be made to extend from the base of the thumb

of the fingers in many instances.[13]

The patient must not bear weight upon the palms while moving in bed. The deformities which occur most commonly in the fingers include ulnar deviation of the fingers producing the flipper hand; flexion contracture of the metacarpophalangeal joints; and interphalangeal flexion contractures; rarely dislocations.

Deformities may be overcome in rare cases by traction. If necessary,

the arthritis has been quiescent for at least three months. A number of operative procedures have been described for deformities of the wrist and hand. The only ones of much practical value are the freeing of contractures, and arthrodeses

Arthritis of the Temporomandibular Joint.—1. Limited motion occurs frequently, but ankylosis is rare.

2.

3.

subsidence of the acute symptoms.

4. The range of motion may be increased by cork or wooden wedges inserted between the teeth. Manipulation under anesthesia is at times helpful.
5. Arthroplasty of the temporomandibular joint may be resorted to in severe cases.

REHABILITATION

If the patient is seriously

be set

f the

about 2 to 3 inches longer. By means of such a baker, any variation in heat from 1 to 4 bulbs may be used.

The bulbs are lighted in sufficient number to produce the desired amount of heat; when this has been obtained, most of the bulbs may be turned off and a small amount of heat will maintain the desired temperature. If desired, the apparatus may be made large enough to include the entire body, except the head. The body baker is preferable to the hot wet pack as the former is simpler to use, and there is not as much initial heat with gradual lowering of the temperature as occurs with the wet pack.

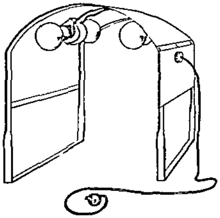


FIG. 147 —Homemade baker (Council on Physical Medicine and Rehabilitation.)

reflector—highly polished

d in multiple with heavy
e above baker is designed
d for the body, supports
should be 2 or 3 inches longer

Baking is a most comfortable way of providing systemic heat. As with other forms of thermotherapy, the *heat tolerance of the patient must not be exceeded* Blistering or burns may develop in sensitive individuals. The cradle supports should be spaced sufficiently wide so as to avoid contact with the skin An average treatment requires about thirty minutes, and may precede daily massage. The patient should be covered with blankets

producing dull red
decreasing with the
concentrated in the
superficial layers of the skin and is slowly ... by the underlying blood. Infrared radiation of short ("near") wave length (7,600 to 15,000 Angstrom units) is tolerated twice as well by the average skin as that of long ("far") wave length (15,000 to 150,000 Å). For clinical use there are advantages inherent in both types However, the amount of irradiation, even with the penetrating wave lengths, which can be tolerated is rather

CHAPTER 21

PHYSICAL THERAPY IN ARTHRITIS

By Morris A Bowie, M.D.

Intelligent *use of physical therapy is one of the most valuable adjuncts in the management of the arthritic patient* Its injudicious use may cause harm.

the past, physical medicine has been looked upon with distrust by many

of fighting [29]

"Physical Medicine includes the employment of the physical and other effective properties of light, heat, cold, water, electricity, massage, manipulation, exercise, and mechanical devices for physical and occupational therapy in the diagnosis or treatment of disease "[42] Physical measures which have been used in the treatment of rheumatic diseases include heat,[8]

application

In older people physical medicine must be used conservatively and in small doses since they may not tolerate heat and other measures as well as younger individuals *Due precautions must be taken with the application of any measure*, and frequent observations made, especially when any form of heat is applied Accidents may occur because of improper technic or inattention by the technician, lack of cooperation or unusual sensitiveness on the part of the patient, or from a faulty piece of apparatus. Most of the accidents for which physicians have been sued are *burns caused by thermal, electrochemical or photochemical energy* although mechanical injuries and even electric shock have been reported [125] When technicians are employed, they should be carefully supervised by the physician in charge The latter is to a certain extent legally responsible for their actions

In order to achieve a really effective therapeutic program for the arthritic patient, physical therapy must be carried out at home for long periods of time.

low.[155,217] Lamps producing a large amount of near infrared energy are the 250 watt CX Mazda bulb, the 250 watt carbon filament bulb, and the newer 250 watt reflector heat lamp in addition to the 500 to 1,500 watt tungsten filament bulbs The shape of the reflector is an important item in

the sensitivity of the part Infrared lamps are the least effective means of

The above methods of applying dry heat all depend for their effectiveness on the emission of infrared rays They are more economical, easier to manipulate, and less dangerous than long or short wave diathermy Direct inspection of the part being treated is always possible. Effectiveness of the treatment may be enhanced or varied by the application of hot wet compresses or counter-irritants. The former may be kept continuously wet by the addition of water to replace that lost by evaporation

This form of treatment should usually be given several times daily, but it may be used at low intensity as a continuous application in some conditions Each period should be at least twenty to thirty minutes in length

burning sensations during treatment indicates that the lamp should be moved farther from the patient.

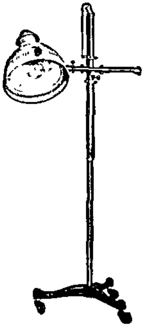


FIG 148 — Large luminous heat generator on stand (Courtesy of The Burdick Corporation)

Electric Cabinet Baths.—These are used only in robust patients Treatment lasts from five to fifteen minutes and is followed by a warm shower and gentle massage or a salt rub. The patient must then lie down and rest for an hour during which time he gradually cools off Such baths may be employed once or twice weekly Their value in chronic arthritis is doubtful, but may afford some relief to the patient with primary fibrositis

Hot Air Devices.—1. **Hot Air Douche.**—One may use an ordinary hair drier to blow a current of hot air against painful joints (such as fingers and hands) for periods of ten to thirty minutes.[129] Penetration of heat in this method is limited.

2. **Heating Jacket** —It is very difficult to heat all surfaces of the shoulder by ordinary means because of its irregular contours In using radiant

32 HELLEBRANDT, F. A.: Jour. Am Med. Assn, 142, 967, 1950.
33 HENDRY, A. M.: Practitioner, 155, 89, 1945.
34. HERBERT, J. J. Revue du Rheumatisme, 18, 369, 1951.
35 ——— Mém Acad Chir. Paris, 77, 645, 1951.
35a HOLLANDER, J. L.. Personal communication
36. HOLMDAHL, D E, and INGELMAK, B. E: Acta. med Scandinav supp, 170 568, 1946.
37. HOWELL, T. H.. Clinics, 4, 1204, 1946
38. HOWORTH, B.: Jour Am Med Assn, 131, 398, 1946.
39. JORDAN, H H.: *New York State Jour. Med., 39, 1823, 1939*
40 IRWIN, C E., and EYLER, D. E: Jour. Bone & Joint Surg, 33, 825, 1951.
41 KAVANAUGH, J. K. New Jersey State Jour. Med, 46, 430, 1949
42 KELLY, L C, New York State Jour of Med, 42, 144, 1942.
43 KEY, J A. Western Jour. Surg, 42, 385, 1944.
44 KESTLER, O C: Geriatrics, 2, 283, 1947.
45 [illegible] Saunders Co, 1941
46
47.
48.
49.
50.
51.
52.
53. ——— Clinics, 4, 1465, 1946.
54.
55 [illegible] 0, 22, 1951
56
57.
58.
59.
60
61. [illegible] Bone & Joint Surg, 31, 805, 1949
62 OSGOOD, R. B: [illegible]
63 PEMBERTON, R [illegible]
64. PEMBERTON, R. [illegible] ment of Chronic Arthri[illegible]
65 PRESTON, R L [illegible] 1937
66 [illegible]
67 [illegible] 238, 142, 1948
68. [illegible] 260, 1939
69. [illegible] E: Jour. Bone and
70 SOLOMON, W. M.. [illegible]
71. [illegible] 131 180 1946
72 [illegible] and End Results,
73
74
75.
76.
77
78
79
80
81.
82
83.
84
85.
86
87. [illegible] 1, 209, 117, 1933
88.

heat positioning of the shoulder is useful, but does not entirely suffice. When an ordinary heat lamp is employed, the rays converge on only one surface so that little heating of the periarticular structures in the axillary *space is obtained.* The usual baker does not produce uniform heating of

ture is controlled by an accurate thermoregulating mechanism.[134] This apparatus permits the application of regulated conductive heat to all surfaces of almost any region of the body, such as the hip, foot, or shoulder without resorting to more expensive apparatus.

Infrared Radiation

Infrared radiation is used chiefly because of its local heating effect. It may be useful in relieving 1 or 2 painful joints in the acute stage of an arthritis or in decreasing pain, stiffness and muscular spasm in joints which are the seat of a chronic rheumatoid process.

It may be given several times a day; the maximum heat which can be endured by the patient is focused ...

In ... the administration of massage.

Electric Blanket.—The commercially available electric blankets do not strictly belong to the armamentarium of the physiatrist, but they do possess usefulness in preventing or ameliorating the morning stiffness of which most arthritic patients complain. Some bedridden patients with an overactive vasomotor system seem to do particularly well under an electric blanket at night or on chilly days. Electric sheets have recently appeared on the market.

Diathermy and Diathermy Machines.—Heat is produced when an electric current is passed through tissues. This action is utilized in diathermy

muscles. The intensity is sufficiently low so that the temperature developed in the tissues is below that which will destroy tissue or impair its vitality. This is the most efficient form of heating the deeper tissues, and is especially

Machines currently available and approved for inclusion in "Apparatus Accepted" employ either an 11 or a 22 meter wave length. The frequencies assigned to medical diathermy and to industry have recently been reallocated so that after June, 1953, all machines now in use must be screened

Physical Medicine in Rheumatoid Arthritis[7,164 170 185 211 212 214]

Objectives of treatment are to relieve symptoms, shorten duration of disease, and prevent or correct deformity The program of physical medicine should be fitted into the patient's total regime Amelioration of the symptoms by the steroid hormones does not invalidate the need for a well chosen schema of physical[190,210] therapy. Hospitalization preliminary to home treatment may be advantageous

1 Acute stage with fever and tender, swollen joints
   [illegible]
   q i d
   f No exercise except passive motion after each application of heat
      (1) Mild faradic stimulation may be used
2 Subacute and chronic stages
   a *Triad of heat, massage, and graded exercises one or more times daily*
   b Systemic heat once daily tub bath, tank, cradle, packs
   c Local heat several times daily infrared, paraffin, contrast baths
   d Massage once daily if feasible Gradually eliminate and rely on exercise
   e Exercises
      (1) Muscle setting, deep breathing, and abdominal retraction hourly × 8
      (2) Graded exercises four times daily assistive at first, then with gravity eliminated, active as soon as possible, and finally progressive resistance exercises
      (3) Use pulley-sling devices when shoulders, hips are involved, other [illegible]
   f [illegible] ...ie session of graded exercises
   g Postural and gait re-education
      (1) Teach proper use of crutch canes, crutches, walker[4 49 54,101 104]
      (2) Correct anomalies of gait by walking in front of mirror
3 [illegible] particular patient
   d Train relative or friend
   e Swimming and underwater exercises when facilities are available
4 Physician's role
   a **Take time to explain in detail the function of physical therapy**
   b Assay patient's performance frequently, otherwise he will think physical treatment is unimportant
   c. **Do not confine ambulatory management to two or three diathermy sessions.**

to prevent interference with other frequency channels[41,76] It is recom-

straight lines and can be reflected, refracted, and absorbed Its physiological effects in some respects resemble those of diathermy but in others resemble those of radiant energy from infrared lamps Its therapeutic effects are simply those of heat liberated in the tissues "[15] The fundamental reason for using currents of high frequency is that the body temperature may be raised without injury or electrical stimulation Because of the deep penetration of these currents, diathermy is the physical agent of choice in any condition where deep heating of the tissues is indicated

Wave lengths and Frequencies of Medical Diathermy[90,156]

	Range		*F C C Bands*	
Diathermy	*Wave length*	*Frequency, megacycles*	*Wave length*	*Frequency, megacycles*
Microwave	1 mm–1 meter	300–300,000	12 2 cm	2,450
Short wave	3–30 meters	10–100	22 0 meters	13 66
			11 0 meters	27 32
			7 3 meters	40 98
Long wave	30–3,000 meters	0 1–10		

painful shoulders, sprains, and for pelvic inflammatory disease It provides more definite localization of high frequency currents than the other two types Dosage is somewhat easier to control with the long wave apparatus, but *actually control of dosage is rather empirical* The milliameter on the machine indicates that current is passing through the apparatus, *but it does not measure the energy passing through the patient.* A comfortable heat sensation of the patient's skin is the only recognized method of regulating dosage at present. Burns are apt to occur unless frequent inspection and careful technique are observed The treated area should be thoroughly inspected *afterward*

In many instances, burns are due to collections of moisture between the skin and the electrodes resulting in a concentration of energy at these points. Some patients are either unaware of the sensation of burning or think that the discomfort goes with the treatment Hence, they should be

formance. Written instructions and class demonstrations have proved feasible and effective at the Mayo Clinic.[219,237] If *sufficiently detailed outlines are given*, a large number of these patients continue treatments at home.[69,117,197,209] Home care programs under the sponsorship of hospitals[24,220] and other agencies include visits by physical and occupational therapists, home care is cheaper than hospitalization. In smaller communities lacking a hospital physical therapy department or a trained therapist, several physicians may collectively invite a trained therapist into the community and refer arthritic patients to the therapist. In strictly rural areas, mobile physical therapy units are possible means of meeting the demands of good care. Such units are employed widely in Great Britain under the new Health Act

Prescription for Physical Medicine[63,154,155,223,235]

1. Specify diagnosis and indicate any *special* instructions or precautions re patient.
2. List the parts or area of body to be treated.
3. [illegible]
4. Number and frequency of treatments during the day.
5. Direct what home measures are to be employed.
   a. Technician to check patient's performance frequently
6. Include date for re-evaluation by physician at which time he should[196,206]
   a. Ascertain that directions are carried out properly;
   b. Grade patient's performance;

TYPES OF PHYSICAL THERAPY EMPLOYED IN ARTHRITIS

I. HEAT

The application of heat or thermotherapy may be directed either locally or systemically. Thermal energy may be transmitted from one body to another *by conduction* (hot packs, hot water bottle, electric pads, hot air blowers or chambers, chemical heat pads, the application of hot solids or semi-solids [paraffin, mud, etc.], hot tub baths, Hubbard tank, whirlpool, and therapeutic pool); *by radiation* (luminous and infrared lamps, bakers, household electric heaters); or *by conversion* (long and short wave diathermy, microwave). The physiological effects from local application of any of these measures differ very little except that penetration is least with conduction and most with conversion methods.

in which *inflammation is present tolerate deep heating less well* than normal tissues.[157] Anesthetic areas and regions should not be treated locally.

Precautionary Measures in Application of Diathermy[32,157,165]

1. Have patient assume comfortable position, either sitting or lying
2. Use wooden table. *Do not employ chairs, tables, or beds with metal parts* ... to ignite the mattress
3. ... s, keys, knives, money, watch,
4.
5. Instruct patient to inform operator of unpleasant sensations
6. Inspect the toweling or interposed material. If this is too wet, replace.
7. *In treating head, therapy must be stopped if untoward symptoms occur*   drowsiness, inability to concentrate, vertigo[215]
8. Limit duration of treatment to 20 to 30 minutes   *Set interval timer at start*
9. WITH MICROWAVE DIATHERMY
   a. Use distance of 5 cm from director to skin and power output of 60 to 80 watts.
   b. Duration of treatment limited to 20 to 30 minutes.[158]
   c. Use with extreme caution over:[139,157,159]
      (1) Any area containing metallic bodies or plates (e g., bone plate),
      (2) Ischemic areas,
      (3) Bony prominences,
      (4) Epiphyseal regions of growing bones (and children under five);
      (5) The eyes,[139,193]
      (6) Edematous tissue
   d. *Observe other precautions as with long or short wave diathermy.*

Short wave diathermy is applied by means of an induction cable which provides an electromagnetic field and yields higher muscle temperatures than is possible with condenser pads or air-spaced electrodes; the latter ...

type. The flexible, heavily insulated cable may be ... to be treated or be placed in the form of a pancake coil. Diathermy has many applications and has enjoyed wide popularity in recent years. Unfortunately in the arthritic patient it is often used as the sole physiothera- ...

patient is concerned.

In arthritic patients with ... *systemic heating is usually indicated*, and, for ... is excellent provided that the physici... a suitable

recovery period General vasodilatation and a rise in the body temperature can be attained within thirty to forty-five minutes by insulating the patient in blankets and placing the cable over or under the patient's back. In fever therapy, the treatment may extend over several hours, in this case a fever cabinet is preferable to the blanket technique. The first few treatments should be short (ten minutes or so) since aggravation of symptoms may be induced.[122 131] *Diathermy is not essential for the proper treatment of rheumatoid arthritis.* It is contraindicated in the acute stages In the

chines. However, diathermy will often induce a good psychic effect on the

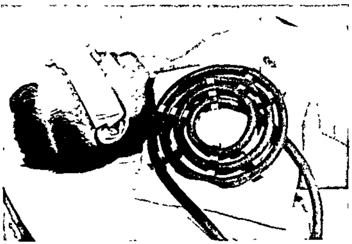


FIG 149—Short wave diathermy to the shoulder Pancake coil technic—electromagnetic induction. (Coulter, courtesy of Med Clin North America)

tions directly onto the surface An output of 60 to 80 watts has been used for most of the experimental work in both animals and man [92 158 159 196] In order to decrease undesirable reflection from the skin, the dielectric *Mycalex* may be interposed between the director and the skin [84 159] A new

detected, and that patients are aware of a pleasant sense of warmth without being conscious of the higher internal temperature [85,106 176 192] In meat, the

while undergoing treatment. The range of motion in a contracted joint can be extended either actively or with assistance while in the water, buoyancy of the latter permits a greater range of motion than would be possible for a stiff joint when out of water.

Although the commercially available types are somewhat expensive, a useful substitute can be assembled by a local plumber at moderate expense Detail scale drawings of a whirlpool bath, luminous baker and other simple

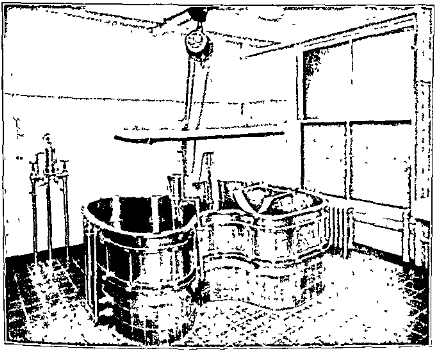


FIG. 155 —Modified Hubbard tank Note overhead pulley and head rest (Currence, courtesy of Arch Phys Ther)

iel suction tee, with an air vent pipe from the tee to the top of the bath to provide adequate air mixing. The inlets are placed 4 and 18 inches above the bottom and near the outlet. Standard 1-inch galvanized pipe is used A 2½-inch overflow outlet is placed at one end near the top to keep the water at a safe level.

Hubbard Tank.—This type of hydrotherapy is especially valuable in arthritis with involvement of multiple joints, contractures of the joints, especially of the hips and shoulders, and in many cases of spondylitis. In addition it is invaluable in many neurological conditions such as poliomyelitis, or spastic paralysis as well as in fractures of the hip and post-operative

rise in temperature at the meat-fat junction is always above that observed in an all meat medium. In an all fat medium, the temperature may rise to twice that in an all lean medium. When bone underlies lean meat at a depth of less than 1 cm., the tissue is heated considerably more than if bone were not present. At distances greater than 1 cm., the temperature gradient approximates that with no bone present. In the living body, the temperature differences will be far less than in dead tissues because of the mediating effect of the circulatory system.[39,67,159] In living tissue the increment of temperature rise is greater in the first ten minutes of radiation.[167] The extent to which blood flow is increased depends on the intensity and duration of heating.[79,114,204,225] Average rise in deep tissue temperature (2 inches)

Medical Diathermy

1 [illegible] f burns.
2 [illegible]
 b Damped waves can be used for electrocoagulation or electrodesiccation.
3 Advantages of microwave:
 a. Affords good selective deep heating to limited areas.
 b. May prove to be superior for treatment of painful shoulder.[238]
4 [illegible]
5 [illegible]
6 [illegible] essential.
7 Consults texts for details of application [15,42,60,122,155,177]

is to about 104° F. and at the bone cortex 6 5° F.[67,68] Intense heating with microwaves may damage growing epiphyses,[67,234] induce cataracts[139,157,159,194] promote degeneration of the testes,[109] cause burns and necrosis of the skin,[16,71] produce focal lesions of the nervous system,[106,174] and give rise to dangerous temperatures adjacent to metal plates.[77] Rises in intra-articular temperature averaged 8.4° F.[101] With due caution and repeated observation of the patient, no harmful results should accrue. *The eyes should be protected.*

Tremendous forces are developed during the propagation of ultrasonic waves.[6,88,99,146,168] These waves are commonly generated by placing a [illegible] electric field in the same direc- [illegible] the same [illegible] produced

treatment of tendon transplants and plastic operations on the joints Weakened muscles and joints can perform motions under water, which would be impossible out of water. The buoyancy of the water largely eliminates the force of gravity When a body is submerged in water, it will lose as much weight as the displaced water weighs. Thus, a patient weighing 132 pounds (60 kg.) will weigh only 6 pounds (2.7 kg.) when lying in water[80] Muscle spasm is decreased, and the enveloping, uniform heat, which is constant throughout the period of treatment, stimulates cutaneous and venous circulation.[173] *Passive or assistive exercises may be carried out and underwater massage may be employed.*



FIG 156.—Modified Hubbard tank. (Currence, courtesy of Arch. Phys Ther)

Hydrotherapy in a Hubbard tank or therapeutic pool should be used *cautiously in acute rheumatoid arthritis because of the probability of fatigue.* The frail patient and those with hypertension may not tolerate even a short session Mild shock is occasionally precipitated in such patients.

In a hydrotherapeutic tank or pool, the temperature of the water is kept between 90° to 96° F. This is usually comfortable to the patient and encourages muscular relaxation *In the therapeutic tank, the temperature of the water may be changed rapidly.* Some patients in the subacute or chronic stages of rheumatoid arthritis may tolerate an initial temperature of 100° F. After five minutes, if no untoward symptoms have occurred, [illegible] 106° F. A rise in [illegible] intained after [illegible] usually best, however, to reduce the water temperature to that of the initial level before removing the patient. Passive, assistive, or active motion of the affected joints should be carried out throughout the treatment period. Treatments may be given every two or three days if no reaction occurs within twenty-four hours. The more robust patient may be treated daily.

Ultrasonic vibrations *induce mechanical, chemical, and thermal effects in various media.* The *destructive action of ultrasound is due to the formation of gas bubbles by cavitation*, cavities or hollows form in a liquid medium due to

Sub-
can be
Some

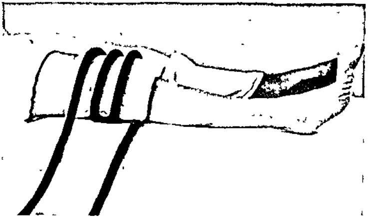


FIG 150 —Short wave diathermy to the knee (Coulter, courtesy of Med Clin North America)

Contraindications to Local Use of Diathermy[29 32 156 215]
1 Acute infectious arthritis and other inflammatory processes (cellulitis, thrombophlebitis, osteomyelitis, tuberculosis)
2 Over ischemic or anesthetic areas (occlusive vascular disease, nerve injuries)
3 Over metallic implants, adhesive or moist dressings
4 Do not use through the pelvis, lower back or abdomen during pregnancy or profuse menstruation
5 Over known or suspected malignancies or metastases
6 Over freshly traumatized tissue, in hemorrhagic diseases, or to the back or abdomen in known peptic ulcer
7 Do not use microwave over joints with effusion or edematous tissue Water and other fluids absorb a high proportion of microwave energy
8 Transcerebral applications should be done only by those familiar with the hazards involved[215]
9 Do not use where simpler methods of applying external heat give good results

solids are disintegrated, and emulsions coagulated The viricidal and bacteriocidal action of ultrasound is due to cavitation *Chemical reactions*

released from the blood[6] *Thermal effects depend on the reflection of waves*

The greatest advantage in underwater therapy for the arthritic patient is in the elimination of the force of gravity by the buoyancy of the water.

effective in stimulating the flabby tone of the latter and in restoring their normal use.[108 129 131 149 150] Lowman[150] in discussing hydrogymnastic technique has emphasized the boost to a patient's morale when he finds that he can readily move his limbs and/or trunk in all planes. Water induces a particular stimulus to motion and induces a pleasurable sense of activity. Stretching or extension of affected joints by the technician may be carried out in comfort under water (as compared with weights and pulleys attached to the bed). Lowman recommends that the technician be especially trained in kinesiology and muscular reeducation.

Hubbard Tank Therapy in Arthritis[108 147 150 173]

1. Water temperature
   a. Initially 96 to 98° F
   b. Raise gradually to 101 to 104° F during first three or four minutes. Use cold head compress if indicated
   c. Lower slowly to initial temperature during latter part of treatment period
2.

   ...weekly, with improvement, treat daily
   e. After removal from tank, dry quickly and wrap in blankets for a half hour to maintain elevated temperature
3. Underwater massage
   a. Effleurage and gentle stroking massage may be applied above and below affected joints. Kneading may also be used
4. Underwater exercises
   a. Passive and assisted active exercises for subacute cases.
   b. Active exercises as soon as permissible; supervision by technician necessary to avoid faulty movement patterns[10]
   c. Add resistive exercises (either manual or mechanical) for weakened muscle groups
   d. Move turbine agitators to position most favorable to aid patient in performing active exercises

Resistive exercises may be employed to advantage in certain cases;

between interfaces, the frequency of the wave length, and the degree of cavitation.

The biological effects have been widely investigated.[23,83,92,93] Various *physiological and pathological changes have been observed.* Fatigue, irritability, deep bone pain[93] have been noted in volunteers subjected to an area in which ultrasonic radiation was active. Hyperemia, edema, vesiculation, and even gangrene of the skin and deeper tissues may occur.[5,7,110] The brain can be damaged with focused ultrasound[152] *The action potentials of the sciatic nerves can be flattened*, and, in some animals necrosis of the cauda equina and myelin degeneration induced[5,235] An abrupt rise of 5 to 10° C. can be attained in bone marrow and cortex after a relatively short exposure at a power output of 5 to 10 watts[169] *Ultrasound is contraindicated* over growing epiphyses, the gravid uterus and reproductive glands, in the region of the cervical sympathetic chain in cardiac patients, and over the extremities in Buerger's and other occlusive vascular diseases. As a diagnostic tool, ultrasonics may prove to be of value in the localization of brain tumors gall stones, foreign bodies, and in outlining the size of the abdominal viscera.[53,151] The effect of this new modality is under active investigation[105] in the field of neoplasms.

European literature has contained enthusiastic accounts of the effect of ultrasonics in a variety of disease states, including many rheumatoid conditions, but much of this has been uncritical Several general reviews may be consulted,[11,100,105,127,168] and a bibliography is available[47] The Council on Physical Medicine and Rehabilitation *has not as yet approved the use of ultrasonics for general therapy.*[40]

Ultrasonic energy is applied by means of a sound head, which is usually immersed, along with the part to be treated, in a container of boiled water. A water pillow, made by filling a condom with boiled water, may be fitted over the sound head; this allows adaptation to irregular contours A distance of an inch or more is maintained between the head and the part to be treated. Most workers have found it desirable to use a *stroking motion rather than a stationary position* in order to avoid overheating of tissues and allow some degree of recovery before the next stroke The head and the part to be treated may be rubbed with aerosol to eliminate air bubbles. The input and output of an ultrasonic generator can be measured easily, but the amount of energy entering the tissues is difficult [illegible] ultrasound may be

[illegible]

3 watts per square centimeter of surface) at a frequency of 800,000 cycles per second, with larger energies the *head must be moved* rhythmically to avoid overheating. Higher frequencie [illegible]
lower ones cause considerable pain.[99]
recommended for general use in the tre[illegible]
other methods of proved merit are available.

B. Forms of Applying Moist Heat

Hot Fomentations.—These may be applied by dipping heavy Turkish towels, blankets, or woolen cloths into very hot water (about 115° F.),

Therapeutic Pools.—These are expensive to install and to operate, but offer the advantage that a more extensive program of hydrogymnastics is possible than with a tank. *Furthermore, reeducation in walking is possible.* The Currence modification of the Hubbard tank allows complete range of motion of all [illegible]
the tank by [illegible]
an overhead [illegible]

[illegible]

in such a tank or even a large bath tub; a bubble bath effect is created when water is forced through *the perforations, and this adds to the bolstering power of the water.*

II. THE USE OF COLD IN ARTHRITIS

The manner of applying heat or cold determines whether the main effects will be mediated by local, systemic, or reflex action.[72] Locally, cold brings about cutaneous vasoconstriction, *restricts* exudation and bleeding, decreases cellular activity, and ameliorates pain by desensitizing nerve end organs. Chilling of the body surface as a whole induces peripheral vasoconstriction, which may also involve the deeper vessels if the exposure is long enough. Capillary permeability is altered (generally reduced)[111] and the blood pressure is often elevated. The latter effect is used as a test for the early diagnosis of hypertension. Leukocytic response is lowered, and *phagocytosis by the fixed tissue cells appears to be impaired.* Prolonged exposure may lead to necrosis of the tissues as a result of thrombosis of the deeper vessels Considerable damage to the nerves may also result The complexity of the subject and the factors involved were seen in the studies of immersion foot during World War II. Previously Fay and Smith[75] had developed systemic hypothermia or artificial hibernation. The anesthetic effects of local refrigeration for devitalized extremities, as originally *proposed by Allen,[2] has found use in a variety of medical and surgical* conditions. Intra-articular temperatures preceding and following various procedures have been determined by Horvath and Hollander.[104] In general the correlation between joint and surface temperatures was good although there were some exceptions. Surface temperature over the knees in the normal subjects ranged from 85° to 89.5° F. and corresponding intra-ar[illegible] temperatures from 88.6° to 91° F. In rheumatoid arthritis a positive [illegible] es and relative clinical [illegible] mal or inflamed joints induced a *drop* in the intra-articular temperature to the extent of 2.2° F. The joint temperature quickly returned to normal after cessation of the packs, and higher values were recorded within ten minutes, indicating a [illegible] intra-articular

wringing dry, and placing over the part to be treated One or more hot water bottles or an electric heating pad may be arranged over and around the compress, which is then covered by a dry blanket or heavy towel A layer of cellophane or waxed paper directly over the compress prevents too rapid loss of heat, and cuts down the frequency of re-application The gradient between the temperature in deep muscle, subcutaneous tissue and skin is decreased in the packed area [217] Changes in local and peripheral temperature and in blood flow indicate that a single application for 30 to 60 minutes is as effective as more frequent changes in packs [129 217] A simple

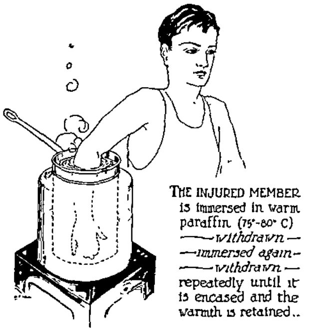


FIG 151 —Warm paraffin bath (Handbook of Physical Therapy, Am Med Assn)

portable device, electrically heated by self-contained thermal units within a moistened pack, may soon be available [91] The treatment period may last for fifteen to sixty minutes and three or more treatments administered daily Hot fomentations are of great value in the treatment of acutely painful, swollen joints The addition of a cupful of magnesium sulphate to the bucket of hot water may produce better symptomatic results and afford mild to moderate analgesia This agent is somewhat drying to the skin, a bland oil or lotion may be rubbed in afterward. Several joints may be conveniently treated at one time A rubber sheet or waxed paper under the compress will protect the bed linen. If joint pain and swelling increase following such applications, the treatment should be discontinued. Hot fomentations are also valuable in the treatment of painful stiff joints in the chronic stage of rheumatoid arthritis. Such moist heat may produce more

effects similar to those noted from the application of cold. Simple passive

normal or rheumatoid joint. Jones had previously demonstrated the importance of synovial fluid as a means of dissipating the heat of friction in the equine stifle joint.[110] More prolonged application of cold than that used by the above investigators has been shown to cause a decrease of approximately 20° F. in the intramuscular temperature [14]

Cold Compresses.—A Turkish towel is immersed in ice water, wrung out, and applied to the part An ice bag may be placed over the compress to accentuate the cooling effect. The very acute stage of bursitis is an indication for the use of cold, since heat may prove to be intolerable. In the acutely inflamed joints of other rheumatic conditions, especially those due

Cold Air.—This may be circulated in a box placed over the inflamed joint [14] Some of the newer hair dryers for home use are designed to deliver both hot and cold air.

Contrast Baths and Scotch Douche.—The former was discussed under the section on Heat. The feet or hands are placed in the hot water for three to four minutes, and then plunged in cold water for a minute or less The temperature extremes of the water should be as great as the patient can tolerate The manoeuver is repeated 5 to 8 times

With the Scotch douche hot and cold water under pressure is alternately sprayed over the patient This may be used as a tonic measure following a hot tub bath. Most patients experience a sense of well-being A simple home spray attachment may be used, and is a fair substitute for the pressure type.

III. MASSAGE IN ARTHRITIS

The use of massage for the relief of human suffering dates back to antiquity Anointing parts, or all, of the body with oil was a common practice During this procedure some stroking of the skin and deeper tissues was carried out Often it was preceded by bathing in hot water or hot mud Its firm scientific basis as one of the cornerstones of physical medicine has been a by-product of the growth and development of physiology and of the two World Wars It is one of the most useful, easily obtained, and frequently neglected methods of therapy in arthritis Electrical apparatus and other devices, whether atomic or otherwise, cannot effectively replace it To confuse massage with the layman's rubbing does a

many occasions in the general practice of medicine in which ability to give massage is a valuable asset.

The physiological effects of massage are mediated through reflex and mechanical reactions. Gentle stroking massage induces muscular relaxa-

dips than from electric pads. It is also (as in the case of contrast showers) an excellent psychologic form of therapy. Paraffin applications are especially useful in the chronic stage of rheumatoid arthritis and in painful Heberden's nodes. They may be used several times daily.

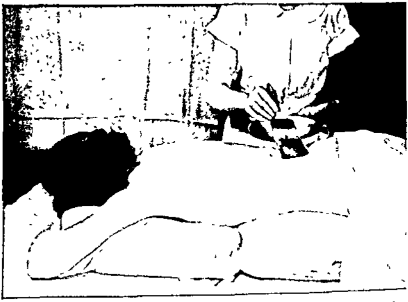


FIG 152.—Method of applying hot paraffin with a paint brush. (Krusen, Physical Therapy in Arthritis, courtesy of P B Hoeber, Inc)

HYDROTHERAPY

Hot Tub Bath.—This is one of the most widely recommended of all measures for general heating in arthritis. It is particularly useful in home treatments. It is one of the most efficient means of elevating the body temperature, and because of this may become hazardous to the patient.[148] The first bath lasts only five to six minutes, primarily to test the tolerance of the patient. Subsequently, the duration of the treatments is increased up to thirty to forty-five minutes if well-tolerated. There must be no undue weakness, circulatory impairment, or exacerbation of joint symptoms. The temperature of the water is gradually raised from 97° to 102° F., or in some cases to 105°, by running hot water into the tub. The temperature of the water should be determined with a bath thermometer. It may be necessary for psychological or other reasons to add magnesium sulphate, sodium chloride (1 pound per 10 gallons of water), or small amounts of pine extract to the bath water. A cool towel around the head lessens discomfort A

sion or tapotement, consisting of hacking, clapping, slapping, tapping, and beating. (*d*) *Vibration.*

Very light stroking or effleurage should be *employed at the beginning of each treatment* [illegible]

[illegible] should not exceed 15 strokes per minute and usually less. The proximal portion is treated first to empty the superficial veins and lymphatics after

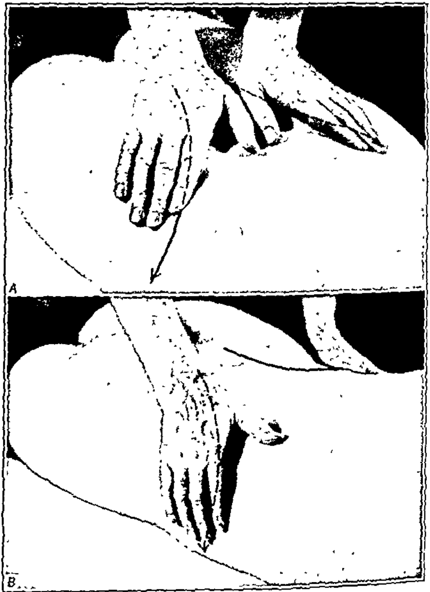


FIG. 158.—*A*, Effleurage of the low back; beginning of motion. Note that the operator's hands are molded to the part being massaged. *B*, End of motion of effleurage of low back begun in *A*.

individual over sixty years of age. Following treatment in the tub, the patient may be sprayed or sponged with tepid or cool water, rubbed down with alcohol, dried and returned to bed. He should be lightly covered and

Technic of Paraffin Application

1. Equipment
   - *a* Double boiler 8 to 12 inches deep or enamel pail 10 inches in diameter
   - *b* *Electric hot plate or stove. Asbestos pad over gas or coal range to reduce fire hazard*
   - *c* Canning grade paraffin and light mineral oil in proportions of four parts former to one part oil
   - *d* Bath thermometer desirable Gauze or roller bandage optional.
   - *e*. Wax paper and flannel
2. Heat slowly with occasional stirring
   - *a* Paraffin-oil mixture melts at 120 to 140° F
   - *b* Remove from flame and allow to cool till a scum forms on top
   - *c* Place receptacle on stool or chair at convenient height
3. Protection of skin
   - *a* Shave hairy parts before application
   - *b* Use cold cream or other lubricant the first few times on parts to be treated
4. For fingers, hand, and wrist
   - *a* Maintain fingers and wrist in neutral position if possible with fingers separated and metacarpo-phalangeal joints in slight degree of flexion.
   - *b* Dip into melted paraffin, allow to remain a few seconds, and withdraw
   - *c* When layer has hardened, repeat the immersion 8 to 10 times (or until the cast is $\frac{1}{4}$ to $\frac{1}{2}$ inch thick) allowing each layer to solidify Fingers and hand must be kept rigid to prevent cracking
   - *d* Wrap in wax paper (or plastic "refrigerator" bag) and cover with flannel
   - *e* *Keep in place for 30 to 120 minutes (Glove cast can be made more rigid by incorporating gauze or roller bandage between one or more layers of paraffin)*
   - *f* Peel off into receptacle Material can be used repeatedly
   - *g* Hand will be red, moist and soft, and in suitable condition for massage, stretching or gentle manipulation
5. For shoulders, elbows, knees, spine
   - *a* Apply with a paint brush (8 to 10 coats) or by means of wide roller gauze impregnated with paraffin
6. Instruct patient to use this form of heat one or more times daily

encouraged to sleep for a short period The effect of heating may be pro-

Prescription for Massage in Arthritis

1. The dosage should be indicated in writing to the technician
   a The time to be spent on each part
   b. Whether massage is to be local or systemic
2 The type of heat to precede treatment should be indicated
3 A statement of type of massage to be given and the purpose to be accomplished should be included.
   a. For stimulation
   b For sedation
4 The diagnosis is important to the technician since it is not within his province to diagnose or prescribe



Fig 159 —*A*, Effleurage of the shoulder and upper arm, illustrating the necessity of even pressure The hand is molded so that it always conforms to the contour of the part over which it is gliding This picture illustrates beginning of this motion *B*, Illustrating end of motion begun in *A* (effleurage of the shoulder and upper arm).

chloride loss; the use of hypotonic (0 3 to 0.6 per cent) saline solutions improves palatability.

Foam Bath.—This is prepared by placing about 10 cc. of saponin solution in 3 to 4 inches of water in a tub. Oxygen or air is then forced into this by means of a perforated pipe or hose. After the foam rises sufficiently (within ten to fifteen minutes), the patient enters the tub. Radiation of heat from the body is restricted by the foam which covers the patient. Some sweating is induced. In nervous patients, such baths have a sedative effect.

Bubble Baths.— [illegible]
spas in various pa[illegible]
must be very fine [illegible]
skin and prevent heat loss. Dilatation of the cutaneous vessels, stimulation of the sensory nerve endings, and stimulation of the respiratory center from increased absorption of the CO_2 are the *chief effects in addition to those of heat.* A carbon dioxide mixing apparatus (the water pressure must be 50 pounds per square inch; a force pump may be added to the system if necessary) may be used to prepare such a bath in the small hospital or at home. Chemical preparations may be added to a tub filled with water at the proper temperature. Four to 8 pounds of table salt are added and mixed with the water, ½ pound of sodium bicarbonate is then stirred in. Finally, tablets of acid sodium phosphate are carefully placed along the sides of the tub. The patient should remain still while in the water in order that the gas bubbles adhere to the skin. A blanket should be placed over the tub to protect the patient from inhalation of escaped gas The [illegible]

Mechanical devices are available commercially to provide an ebullition of fine bubbles through numerous perforations in a rubber covered metal plate

The Full Wet Pack.—This type of hydrotherapy is useful in those patients with subacute or chronic arthritis who should not or cannot be moved into a tub Muscular relaxation and relief of discomfort usually follow this procedure. It is also of aid as a preliminary measure for general or local massage. It requires no expensive equipment or special training on the part of the operator. The whole procedure can be performed by one individual. The method of application is as follows:

The room must be warm (72°). Rubber sheeting is placed over the entire bed, and an old blanket on top of this. A sheet is folded lengthwise in plait-like folds about eight inches wide so that it may be tucked around the patient quickly.

Dip the sheet into hot water (110° F.) and wring out. Quickly spread this sheet under the patient (who is nude). With his arms elevated, one-half of [illegible]

which the masseur lengthens his stroke to include both distal and proximal portions.

For a deeper effect on the underlying tissues and muscles, light, medium, or heavy massage may be used. In *light massage* a force slightly greater than 10 mm. of mercury is applied. The experience born of practice and a



FIG. 160 —*A*, Massage of the ankle joint; it is very important that the movements be performed around (instead of over) the malleolus Pressure is exerted by the thumb or thenar eminence This figure illustrates beginning of the movement *B* indicates end of movement begun in *A* massage of the ankle. The thumb goes around, not over, the malleolus; pressure is not exerted upon the malleolus.

One or two additional blankets may be added to maintain comfort and increase the insulating effect of the covering A layer of warm moist air collects between the patient's skin and the sheet. Rarely is elevation of the body temperature produced. This procedure may be continued for thirty to sixty minutes several times weekly.

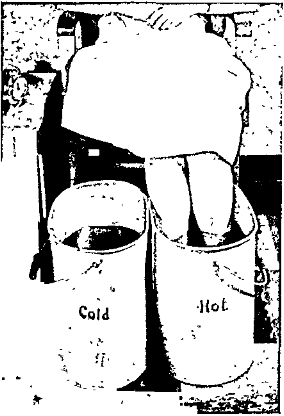
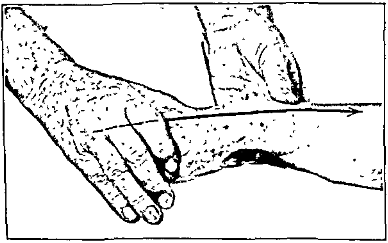


FIG 153 —Contrast baths to lower extremities (Krusen, Physical Therapy in Arthritis, courtesy of P B Hoeber, Inc)

Contrast Baths.—Immersion of the extremities in hot and cold water *alternately is often of value, especially in smoldering rheumatoid arthritis* with considerable vasospasm. A spray or douche can be substituted if the patient can be moved to a tub It is preferable to treat both upper and lower extremities simultaneously if feasible since the increase in blood flow and [illegible] treatment of arms and [illegible] An initial ten minute [illegible] followed by one in cold water (at 60° F.) for one minute; the cycle is repeated, alternating in the hot and cold water for four and one minutes respectively for a half hour.



Fig 161 —Effleurage of a finger Stroking is performed by the operator's forefinger 1 one side of the digit and the thumb on the other The operator's other hand supports e arm and hand



ig 162 —Effleurage of the hand The operator molds the hand to the part and supports the extremity with his other hand

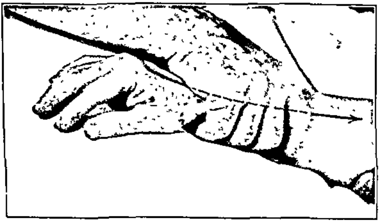


Fig 163.—Effleurage of the hand and forearm

Treatment should be ended by immersion in hot water.[68,82] Occasionally more analgesia is obtained than after the use of dry heat.

Whirlpool Bath.—This hydrotherapeutic bath [illegible]

p

el

fortable to the average patient. The water may be agitated by means of an ejector, a turbine, or compressed air. Well designed turbine agitators, which recirculate the water, are commercially available; ejector types require a thermostatic valve and pressure gauge for regulation of the temperature and force of current. Relatively large volumes of hot water may be

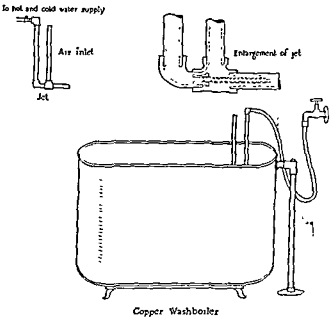


FIG 154 —Homemade whirlpool bath (Council on Physical Medicine and Rehabilitation of the American Medical Association)

used by the latter type Longer models which allow immersion to the waist have recently been marketed. The initial temperature of 96° F. (35.5° C.) is gradually raised to the point of the patient's tolerance for [illegible]

i

to 4.5 atmospheres can be secured by altering the design of the jet and the pumping force, and used as a type of underwater massage[145]

Treatments last twenty to thirty minutes and are administered once or twice daily. These baths are useful in circulatory, traumatic, and chronic rheumatoid conditions. These baths are useful in circulatory, traumatic, and chronic rheumatoid conditions of the upper and lower extremities The combined effect of the high temperature and motion of the water induces a marked stimulation to the circulation. The skin becomes red and warm and the tissues feel relaxed and supple. Exercises can be performed

delicate sense of touch will guide the technician in the amount of pressure he uses to compress any structure or to empty the vessels (one may visualize



FIG 164 —*A*, Effleurage of the neck This figure illustrates beginning of movement, which is seen almost completed in next figure *B*, End of movement of effleurage of the neck, begun in *A*

moment though the stroke may include the length of an extremity or part without a break By this maneuver, the muscles and tissues are squeezed and to a certain extent stretched. The end of the stroke terminates abruptly, and the stroke is repeated in slow rhythmic fashion. The expert masseur will use the palm of his hands for the application of the main

force; thumbs and fingers are molded to the configuration of the part being massaged.

Kneading consists of wringing or rolling movements in which the flat of the hand is employed. In *pétrissage*, the muscles are alternately grasped and squeezed between the hands and fingers. Friction movements utilize a small, circular motion transmitted through the finger tips without lubrication. Too much force is to be avoided with any of the above movements, which can be very forceful. If pain is present during treatment, or muscle soreness persists through the following day, the technician should be notified. Deep massage and friction are commonly employed in the treatment of fibrositis.

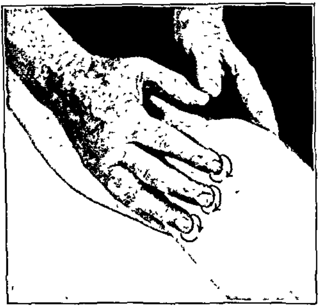


FIG. 165.—Frictions around the knee joint. Arrows illustrate small circular movements with the finger-tips. After frictions are performed in this position, the fingers are moved slightly and the motions repeated, etc., around the patella.

Technic of Massage

1. Pressure is applied perpendicularly to the part in the direction of the long axis.
2. *Never produce pain.*
3. Treatment should start proximally (movements directed centripetally).
   a. To empty venous and lymphatic channels.
   [illegible]

[illegible] comfortable [illegible]

[illegible] rly, and treatment modified accordingly.

procedure because of the possibility of producing fractures of the weakened bones and the ankylosis which sometimes results [125, 153]

Muscular exercises should be performed slowly because rapid motion [illegible] result in [illegible] causing [illegible] skeletal levers, which increase the power and permit rotation. *Muscular action* [illegible]

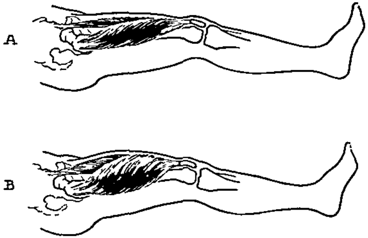


FIG 172 —*Muscle setting* *A* shows the quadriceps muscle at rest *B* shows this muscle contracted (or set) This is performed with the leg extended The position (origin and insertion) of the muscle is explained to the patient He usually learns to "set" the quadriceps by attempting to "squeeze" the muscles of the mid-thigh In some patients this is learned best by pushing the knee backward against the bed

strains and stresses, and subluxations would occur. Elasticity and extensibility are two important physical properties of muscle When a muscle is stretched, it is capable of being extended These properties permit smoothness of action and aid in relieving a sudden impact which might follow the inertia of load, preventing rupture of muscle fibers [26, 61, 62, 64, 65] Contractility is a complex phenomenon; the most powerful contractions occur when the muscle is at its maximal length since its tension is greatest in this position.[64, 224] Muscular tone is increased by muscular exercise. Complete muscular relaxation occurs during life only under deep anesthesia It is the quality whereby muscle remains under sustained tension Lack of muscular tone may add to the instability of joints and allow further injury or strain *Muscle setting and active exercises increase muscle tone* Reciprocal innervation and inhibition of muscles are most important in promoting coordination *Bimanual exercise improves the work capacity of a unilaterally weak muscle group.*[95] Shortening, lengthening, and static contractions as well as the guiding action of muscles influence the degree of coordination Contractures in rheumatoid arthritis may lead to disturb-

Percussion and vibration are not commonly used in physical medicine. These movements are the stock in trade of the athletic trainer and bath house attendant. However, vibratory movement applied with the finger tips and some of the less intense percussion movements may be used briefly at the end of a treatment. *Mechanical vibrators are not recommended;*

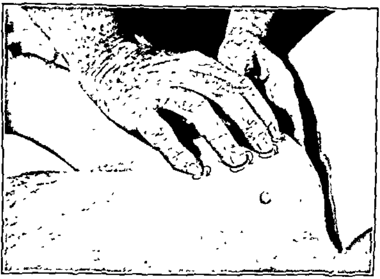


FIG 166 —Frictions at the shoulder, performed with both hands simultaneously

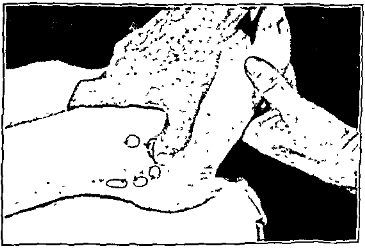


FIG 167 —Friction (circular movements) of the thumb around the external malleolus

their use discourages volitional effort on the part of the patient, and the apparatus may be dangerous [33]

Preparatory to massage, the patient is placed in a comfortable position conducive to relaxation. Position will vary depending on the part to be treated. Tenseness and discomfort interfere with the technical perform-

ances of posture, especially if there is flexion of the knees or hips. Minor flexion deformities of the knees throw the center of gravity backward requiring the spinal column and trunk to compensate by assuming a more forward position.

Therapeutic exercises should be taught to every arthritic patient. In some cases *only* neuromuscular re-education is essential; in others re-development of strength, endurance, coordination and speed may be desirable. Improvement in postural mechanics is almost invariably indicated along with instruction in stretching, relaxation, and breathing. Occupational and diversional procedures may be used to complement any of the above purposes. Exercises are frequently employed with other measures such as hydrotherapy, electrical stimulation, and cervical neck traction. The prescription for therapeutic exercises should be carefully written out, and modified as progress occurs.

Prescription for Therapeutic Exercise[52, 73, 95, 155]

1. Furnish diagnosis
2. List precautions after thorough physical evaluation of patient:
   a Indicate cardiovascular status and make notation of blood pressure
   b Describe defects of abdominal wall, herniae, etc
   c Offer estimate of patient's physical reserve
3. Signify types and purposes of exercise
   a Passive, assistive, free active, or resistive
   b Whether for muscle strengthening, re-education, relaxation, or coordination
   c State which muscles are to be treated
   d Enumerate joints in which increased range of motion is desired
   e If apparatus is to be used, so indicate
   f Suggest supervision of ambulation with crutches, canes, or walker.
4. Designate.
   a. Daily and weekly frequency of exercise sessions
   b Number of sets of exercises and repetitions per set
   c Modifications as progress occurs
   d. Specific list of exercises to be done
   e Whether to be done uni- or bi-laterally
   f. Date on which physician is to re-evaluate

Progressive resistance exercises can be used widely in arthritis These are [illegible] muscle [illegible] "*load* [illegible] "load [illegible] each muscle or muscle group can lift ten times (10-RM) is determined, and, as muscle power improves, the ten-repetition maximum is increased[52, 53, 210] In load-assisting exercise, *the RM means the least amount of counterbalance* help necessary to [illegible] the load-resisting [illegible] times (10 repetiti[illegible] might include one to four sets, each to be done ten times or more; in order to allow for a "*warm-up*" of the muscle, one-half of the 10 RM is used for

ance of massage and limit the beneficial effects to be derived therefrom. *Heat is usually given first. Too much heat and too little massage is not desirable,* and promotes dissatisfaction on the part of the patient. Talcum powder, mineral oil, or a non-viscous ointment may be employed, depending

General Regimen of Massage for the Patient With Rheumatoid Arthritis

1. *Acute stage:* No massage
2. *Subacute stage:* Begin with gentle stroking and later use deeper stroking motions, kneading and friction of the surrounding muscles for ten minutes twice a day, superficial very light stroking may be begun over the joint.[9] This is best performed after at least fifteen minutes of heat to the tissues. **Never cause pain.** The time required for such massage is approximately from five to ten minutes for an arm, ten minutes for a leg, and from ten to fifteen minutes for the back. If multiple joints are involved, all of the areas may be included in the massage. **Always give too little, rather than too much massage.** In involvement of the arm, massage should be begun from the elbow and extend toward the shoulder, then from the wrist to the elbow, and last, from the finger tips to the wrist. If the leg is involved, massage begins from the knee toward the hip to open the vascular channels, then from the ankle to the knee, and finally from the toes to the ankle. In lesions of the back, begin the massage on the sides of the spinal column from the buttocks toward the neck, and then from the buttocks toward the axilla
3. *Chronic stage:* As in subacute arthritis

Pointers on Massage[21,33,43,44,160,161,16*,163,199,2*1]

1. Avoid chilling the patient. Keep the room temperature at 70°
2. Expose only the part being treated. Cover the remainder of the body with a sheet or blanket
3. Both patient and operator should be in comfortable positions. The patient should usually be recumbent to take advantage of gravity in aiding venous and lymphatic flow
4. The part must be completely relaxed, otherwise massage is without value. Remove constricting bands or clothing[33]
5. If hairy parts are to be massaged often, the skin should be shaved
6. Heat should usually precede massage
7. Slow, rhythmic stroking in a centripetal direction is the most useful movement in arthritis[9,163]
8. *Do not massage painful or swollen joints*
9. Massage in arthritis should never produce pain which persists for several hours
10. One of the best ways to acquire a proper technique for massage is to have several treatments from an expert masseur
11. It is almost impossible to do adequate self-massage
12. Observe precautions in the ordering and performance of massage and other physical therapy so as to avoid legal action

the first ten lifts in load-resisting, and twice the repetition minimum in load-assisting. Advantage can be taken of the pull exerted by gravity for weakened muscles The average case of rheumatoid arthritis can tolerate a program of resistance exercises without exacerbation or flare-up if judiciously handled, and especially if concurrent hormone therapy is employed.

Exercises for the Arthritic Patient in Bed

1 Determine rest-exercise balance and work out time log for patient
2 Have patient do deep breathing, abdominal retraction, and muscle setting exercises for the quadriceps, gluteals, calves, forearms 8 to 10 times hourly while awake.
3 In acutely inflamed joints precede passive exercise by use of hot fomentations or packs
4 Start active exercises as soon as possible Underwater and sling suspension exercises, movement with gravity eliminated, and assisted active exercise are usually tolerated early
5 Add occupational therapy of the metric, then kinetic type, adapted to the patient's needs and aptitudes
6 [illegible]
   b Utilize progressive resistance technics early. In these heavy resistance and low repetition induce return of power and bulk of weakened muscle groups [42,53,155,230,240]
7 Supervise ambulation via crutches, canes or walker in order to maintain a good gait or correct a faulty one
8 Modify if overfatigue occurs or pain and stiffness persist.

[illegible]

lack of surveillance by the physician or technician, or from lack of cooperation on the part of the patient. A well adjusted program of exercises, [illegible]

[illegible]

performance indicates a change in directions. The repetitions may be reduced as well as the frequency. An overhead frame with elastic loops for [illegible]

[illegible]

order that the patient perform exercises with precision and with maximum benefit.

Percussion and vibration are not commonly used in physical medicine. These movements are the stock in trade of the athletic trainer and bath house attendant. However, vibratory movement applied with the finger tips and some of the less intense percussion movements may be used briefly at the end of a treatment. *Mechanical vibrators are not recommended;*

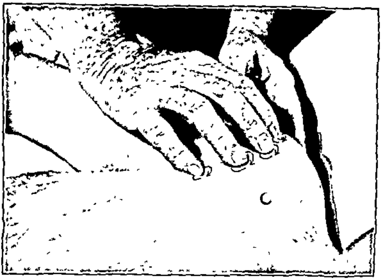


FIG 166.—Frictions at the shoulder, performed with both hands simultaneously

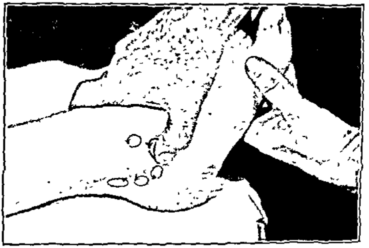


FIG. 167 —Friction (circular movements) of the thumb around the external malleolus

their use discourages volitional effort on the part of the patient, and the apparatus may be dangerous [33]

Preparatory to massage, the patient is placed in a comfortable position conducive to relaxation Position will vary depending on the part to be treated. Tenseness and discomfort interfere with the technical perform-

Exercises for the Arthritic Patient

For the Neck and Shoulders

1. Head hyperextended over the edge of the bed. Tighten the sternomastoids and touch chin to breastbone.
   a. Head hyperextended, fingers locked behind head, elbows back Push neck forward with hands and resist with neck. Push back with neck and resist with hands to starting position.
2. Head hyperextended Rotate head toward either shoulder with sternomastoid taut.
3. Perform circular rotation with the neck
4. Head flat on bed, fingers locked behind head. Bring arms forward till forearms are touching sides of head.
5 Arms outstretched at right angles to trunk, palms up. Touch finger tips to shoulders, return to starting position and repeat. (A small weight in either hand may be used to increase extension of the elbow when contracture is present)
6 Arms outstretched above the body at eye level, palms outward. Bring down to bed level, repeat
7. Arms at sides, palms toward thighs Raise straight above head and touch thumbs to bed above head.
8 Arms at sides, palms toward thighs Bring arms above head, keeping them lifted a few degrees off the bed.

For the Forearms and Hands

9. Forearms at right angles to trunk. Supinate and pronate smartly, keeping the wrists and fingers in extension.
10 Forearms on bed, hands over the edge, palm down Keep fingers in extension Dorsiflex wrist, flex, and repeat.
11. Same position as 10 Close fingers into a fist, and then straighten as much as possible.
12 Same position Invert and evert the wrist.
13. Same position. Approximate fingers and thumb, then spread apart
14 Forearms on bed, hands over the edge, palms up. Repeat exercises 10, 11, 12, and 13
15. Touch thumb to tips of fingers and touch tips of 2-3-4-5 fingers to the thenar eminence instead of the thumb.

For the Chest and Abdomen

16. Breathe deeply, elevating the chest as much as possible. Pull in the lower abdomen with expiration
17. Breathe deeply and hold thorax in full inspiration Then tighten the abdominal muscles and expire against their tension, keeping the thoracic position of inspiration as long as possible. (The use of a small pillow in both supine and prone positions is desirable when this exercise is used in spondylitis)
18 Take a deep breath and hold. Spread the thorax by pulling up along the lower costal border
19. Alternately pull in and blow out the abdominal wall without relation to breathing.
20. Raise head and shoulders off the bed

[illegible] eficial effects to be derived therefrom [illegible] *h heat and too little massage is not desir-* [illegible] on the part of the patient. Talcum powder, mineral oil, or a non-viscous ointment may be employed, depending

General Regimen of Massage for the Patient With Rheumatoid Arthritis

1. *Acute stage:* No massage
2. *Subacute stage* Begin with gentle stroking and later use [illegible] tissues **Never cause pain.** The time required for such massage is approximately from five to ten minutes for an arm, ten minutes for a leg, and from ten to fifteen minutes for the back. If multiple joints are involved, all of the areas may be included in the massage Always give too little, rather than too much massage. *In involvement of* the arm, massage should be begun from the elbow and extend toward the shoulder, then from the wrist to the elbow, and last, from the finger tips to the wrist If the leg is involved, massage begins from the knee toward the hip to open the vascular channels, then from the ankle to the knee, and finally from the toes to the ankle In lesions of the back, begin the massage on the sides of the spinal column from the buttocks toward the neck, and then from the buttocks toward the axilla
3. *Chronic stage* As in subacute arthritis

Pointers on Massage[71, 33, 43, 44, 160, 161, 162, 163, 199, 221]

1. Avoid chilling the patient Keep the room temperature at 70°
2. Expose only the part being treated. Cover the remainder of the body with a sheet or blanket
3. Both patient and operator should be in comfortable positions The patient should usually be recumbent to take advantage of gravity in aiding venous and lymphatic flow
4. The part must be completely relaxed, otherwise massage is without value Remove constricting bands or clothing[33]
5. If hairy parts are to be massaged often, the skin should be shaved
6. Heat should usually precede massage
7. Slow, rhythmic stroking in a centripetal direction is the most useful movement in arthritis[9, 163]
8. *Do not massage painful or swollen joints*
9. Massage in arthritis should never produce pain which persists for several hours
10. One of the best ways to acquire a proper technique for massage is to have several treatments from an expert masseur.
11. It is almost impossible to do adequate self-massage
12. Observe precautions in the ordering and performance of massage and other physical therapy so as to avoid legal action

Exercises for the Arthritic Patient (*Continued*)

For the Hips and Knees

straight with thigh Lower slowly.

24 Heel about 4 inches above the bed with knee straight Move leg laterally and return across the other leg in a scissors fashion
25. Leg straight Tighten the thigh (quadriceps) muscle, relax, repeat
26 Prone position Raise the leg with knee straight Lower slowly
27. Prone position. Bend the lower leg and try to touch heel to buttocks

For the Ankle and Foot

28 Supine position Flex and extend the ankle, curling toes downward and upward with each motion
29. Turn the ankle inward, then outward
30. Rotate the ankle in a circle, curling toes upward and downward

For the Back

31 Prone position Tighten buttock muscles, hold a few seconds, relax
32 Prone position Head and chest flat Raise legs with knees straight
33 Prone position Hands on the buttocks Raise the head and shoulders and hold two to three seconds
34 Prone position Combine exercises 32 and 33 This is a "Jack-knife" exercise
35 Prone position. Fingers behind the head, elbows back Perform exercise 33.

Resistance Exercise for the Knees

36. Sitting with legs over bed Tie sandbag with 5 to 15 pounds of sand (amount physician directs) to the ankle Raise lower leg to extended position and hold for three to five seconds. Lower slowly. Add more sand to the bag as muscle strength improves

Instructions to the Patient

A The exercises circled are especially indicated in your case
B. Each exercise should be performed initially 3 to 5 times on four different occasions daily As you progress, the number of repetitions will be increased to 10 to 20 They should not be done to the point of undue fatigue, nor should they cause pain which persists You should report to your physician if either of these occurs
C The value of the exercises will be greater if you learn to resist each motion with your antagonist muscles
D Exercises 16, 19, 25, and 31 should be done 5 to 10 times each hour in those patients confined to bed.
E Each exercise should be done with precision. Wiggling and purposeless movements are not effective These exercises should accomplish several features.
   (1) They will improve the tone of your muscles and help to prevent muscular atrophy.
   (2) They will accelerate the movement of venous blood and stagnant lymph.
   (3) They will help to reduce the swelling of your joints and to re-establish normal ranges of motion
   (4) They will make you feel better and help to relieve your aching.
F. The exercises as described are designed to be done in bed, but may be adapted to the standing position in most instances with a little re-orientation of the starting position.

on the technician's preference Too much lubrication should be avoided since some degree of friction is necessary for good massage. These adjuvants serve the additional purpose of increasing the operator's sense of touch. Changes in the subcutaneous tissues and muscles not readily felt otherwise can be detected by the skilled masseur. Passive, assistive, active, or resistive exercise may be undertaken following massage and under the



FIG. 1 ... cervical spine, slightly lateral t... the mus... circles

supervision of the technician The amount and time spent must be gauged individually. The treatment period should end with rest or sleep for the patient.

Stage at Which Massage Should be Begun in Rheumatoid Arthritis.—Massage should be started as soon as the acute process in the joints has subsided. It may be given very early in the disease to the adjacent struc-

When the arthritic patient is confined to bed, he sould be placed on a firm, *non-sagging mattress and should practice postural exercises* three times a day. The pillow is removed from under his head and the patient lies in a horizontal position of hyperextension with the hands placed behind the neck or head, if possible, to help raise the ribs and flatten the dorsal spine. A small pillow may be placed under the chest in the beginning. Following thirty minutes in this position, the patient lies on his abdomen in the face prone position for fifteen to thirty minutes, with a pillow placed lenthwise under the chest and abdomen This helps to counteract the gravitational organs. These two positions have been emphasized as the ones which give the nearest approach to complete physiologic relaxation, resulting in elevation of the diaphragm, more active circulation through the vascular channels in the abdomen, increased range of respiratory movements and a greater vital capacity. **The arthritic patient should not spend many hours sitting in a chair** as this permits the affected joints to remain for long periods in positions of the common flexion adduction deformities.

particular attention to those muscles which combat deformity:—the extensors, abductors, and external rotators. Muscle setting may be started with

tients with ulnar deviation of the fingers, a medium weight leather glove into the heel and finger tips of which are sewn moderately stout rubber bands, *will aid in correcting the interosseous muscle atrophy* responsible for the deformity. Wearing these the patient extends the fingers against the pull of the bands.[207] A plastic knuckle support or cock-up splint will hold the fingers in good position between exercise periods and at night.[4]

In rheumatoid spondylitis, special emphasis must be paid to deep breathing exercises since flattening of the chest and atrophy of the thoracic

of the fingers in the forward bending position. Older *patients with degenerative spondylitis* cannot be pushed as hard, but profit by doing exercises regularly

In cervical arthritis of either rheumatoid or degenerative type the Sayre

tures and periarticular tissues rather than to the joints themselves. For example, massage may be instituted within several weeks after the onset of an acute arthritis requiring rest in bed. If the knees are involved, the technician is directed to massage the thighs and lower legs. As soon as the



Fig 169 —Frictions at the lumbosacral joint for arthritis or fibrositis This is one of the important movements in massage of a joint Friction should always be followed by effleurage

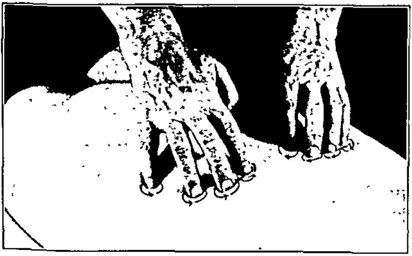


Fig 170 —Bilateral frictions to the low back

acute inflammatory process has subsided, the periarticular tissues may tolerate effleurage, which should hasten absorption of edema.

In the *chronic*

proximal and dis

out over the join

this procedure at home, using enough pull to just lift the buttocks off a chair. A spring scale can be incorporated into the free end of the rope or cord, which is then anchored to a hook or wall stay. With this addition a known pull of 5 to 20 pounds may be maintained for ten to twenty minutes without diverting the patient's attention from exercise. Cervical traction may be used for longer periods once or oftener daily with the patient in the horizontal position, the head of the bed may be elevated 4 to 6 inches to facilitate pull.[195,196]

In **degenerative joint disease affecting the cervical spine**, the pain is usually mainly mechanical with narrowing of the intervertebral spaces (through which the spinal nerves pass) and limitation of the range of motion of the neck In the early stages of the disease, pain occurs only with certain movements of the head and neck and is often more pronounced on one side than the other, the pain later becomes radicular with a segmental distribution along the distribution of the cervical nerves. This picture is often confused with neuritis, bursitis, myositis, etc.

In **degenerative joint disease of the lumbar spine**, exercise may aggravate the symptoms in structurally weak backs. In these individuals, it is often advisable to limit motions by curtailing such activities as golf which results in torsion strain to the lower dorsal spine and lumbar muscles. If there is marked thinning of a disk or marked hypertrophic changes, passive manipulations of the lumbar spine and straight leg raising exercises must be forbidden.

fited by a *firm mattress*, with fracture boards under this, *heat* to the back, *massage*, *muscle setting exercises* and later proper active exercises.

In patients with **involvement of the knees and ankles**, every step must be taken to prevent the possibility of invalidism If these individuals are greatly overweight, this should be reduced to lessen the load upon the lower limbs The early use of *crutches* will assist in weight bearing in these individuals and should be used unless there is an associated arthritis of the hands, wrists and elbows. *It is often difficult to persuade patients with mild synovial irritation to use crutches, but they are extremely valuable if used early*

which is inadvisable

When the **hip joints are involved in a degenerative process**, *diathermy* often affords marked relief if it is applied directly through the hip joint In the *early stages of degenerative hip joint disease*, the patient will usually be benefited symptomatic[illegible] judic
use of diathermy and [illegible] ve
massage, especially of [illegible]
be performed several t[illegible]
much motion as is possible, without pain by passive stretchi[illegible]
joint and fascia of the thigh.[122]

Therapeutic Value of Massage

1. The blood supply to the joints is increased.
2. Lymphatic and venous return are accelerated.
3. Noxious and waste products are removed from the region of the involved joints more rapidly and swelling is decreased.
4. Muscular relaxation and sedation are induced
5. Massage helps prevent fibrosis and adhesions.

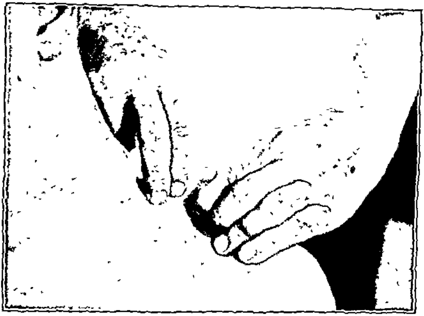


FIG 171 —Pinching pétrissage: the soft tissues are being picked up by one hand of the operator while the fingers of the other hand perform circular movements of the raised soft tissues This is especially valuable in *fibrositis*

Indications for Massage in Rheumatoid Arthritis

Unless contraindicated, every patient with non-acute rheumatoid arthritis deserves proper massage.

Contraindications to Massage in Rheumatoid Arthritis

1. An acutely inflamed joint.
2. Temperature above 100° F.
3. [illegible]
4. [illegible] ngitis.
5. [illegible] *more than two hours) following massage.*
6. Acute burns or acute dermatitis.
7. [illegible] ease is present.

When the arthritic patient is confined to bed, he sould be placed on a firm, non-sagging mattress and should practice *postural exercises* three times a day. The pillow is removed from under his head and the patient lies in a horizontal position of hyperextension with the hands placed behind the neck or head, if possible, to help raise the ribs and flatten the dorsal spine.

organs. These two positions have been emphasized as the ones which give the nearest approach to complete physiologic relaxation, resulting in elevation of the diaphragm, more active circulation through the vascular channels in the abdomen, increased range of respiratory movements and a greater vital capacity. The arthritic patient should not spend many hours sitting in a chair as this permits the affected joints to remain for long periods in positions of the common flexion adduction deformities.

In the patient with an acute arthritis, muscle setting exercises may be started quite early (in pyogenic or tuberculous joint disease this is not true). They should be increased in frequency and duration as tolerated, paying particular attention to those muscles which combat deformity:—the extensors, abductors, and external rotators. Muscle setting may be started with a few contractions each hour and increased to five minutes or more per hour as long as they do not cause pain or undue fatigue. They should be followed in time by assistive and eventually active exercise.

In the chronic stage of rheumatoid arthritis, heat and massage may precede one exercise period daily. It may be desirable to curtail the latter if the patient is tired. *A time log* for the patient will materially aid the patient in keeping to a schedule of meals, rest, exercise, and diversion. If worn, rest splints or braces may be removed for all physical measures. For those patients with ulnar d

into the heel and

bands, will aid in

for the deformity

pull of the bands

the fingers in good position between exercise periods and at night.[4]

In rheumatoid spondylitis, special emphasis must be paid to deep breathing exercises since flattening of the chest and atrophy of the thoracic

generative spondylitis cannot be pushed as hard, but profit by doing exercises regularly.

boggy synovia and occluded lymph channels offers an opportunity for such carefully done massage.

Training of a relative or friend in the rudiments of massage is sometimes indicated when skilled technical help is not available After explanation and demonstration, the trainee must be observed in application; many repetitions may be necessary. Under such circumstances written instructions are a sine qua non.

IV. REST AND EXERCISE IN ARTHRITIS

muscle reeducation [107]

The program for home treatment of the arthritic patient should include exercise periods. Convincing the average patient to take exercises 2 to 4 times daily will require careful explanation, repeated re-evaluations of the patient's technique, and periodic check-up examinations by the physician.

of person. The set-up is inefficient Their muscles are poor, their thoracic cages are narrowed, their diaphragmatic excursion is small, their abdominal viscera are sagged, the weight-bearing lines of their joints are not true,

harder, but, fortunately, there is usually less remodeling to be done."[171]

The above statement is based on the experience over many years of a famous American orthopedic surgeon. It is not overenthusiastic.

Rest in Arthritis.—The above quotation emphasizes one of the most troublesome problems in the arthritic patient, namely, fatigue. In any consideration of exercise, it should be remembered that rest is an essential

fell asleep,

Prescription for Ultraviolet
1. The spectral characteristics of the lamp should be known.
2 The time is adjusted so as to produce a sub-erythemal, minimal erythemal, or first degree erythemal dose depending on the indication for the condition to be treated.
3 Specific written directions to the technician should indicate the dosage, the increment in dosage with successive applications, the frequency per week, and the length of time ultraviolet is to be given.
4. The patient's diagnosis is recorded.

The chief physiological effect of interest in use of ultraviolet for arthritis is that on the provitamins in the skin, which are converted into vitamin D Beneficial effects on some metabolic processes occur, and it is thought that anemia may be materially improved [144] There is no substantial evidence that a "tonic" effect may be derived from ultraviolet, but many physicians accept this as clinically proved Albumin is precipitated, and it is for this reason that the eyes must be protected lest cataracts develop Conjunctivitis develops after intense exposure, as in the familiar "snow blindness"

The therapeutic or middle ultraviolet wave length ranges from 2700 to 3200 angstroms [3000 Å = 0.3 micron (micron = 0 001 mm) = 300 millimicrons] with a peak emission at 2967 angstroms. The penetration is superficial, amounting to 0 1 to 0 5 mm

The use of ultraviolet or sunlight is often of distinct value to the patient with chronic rheumatoid arthritis. A decrease in pain and muscle spasm and a general tonic effect may follow exposure to direct sunlight, or to other ultraviolet radiation. *Some rheumatologists have cautioned against the use of heliotherapy in the acute stage of the disease or in the presence of fever. Others have extolled the value of heliotherapy early in rheumatic joint*

radiation has been advised in this connection The erythema produced by

quartz lamps, the low pressure mercury arcs, cold quartz lamps and electrodeless induction lamps. The closest approach to sunlight is the carbon arc, which is the hottest artificial source of radiation. In the use of artificial heliotherapy in rheumatoid arthritis, the carbon arc irradiation is probably

vantages of this type of exercise over "free" active motion: (1) single muscle groups can be made to function to an optimal degree due to relaxation of the prime movers and lessened resistance; (2) there seems to be a decrease in protective spasm; and (3) the work done by the muscles can be graduated to any amount desirable.[73] DeLorme has thrown new light

with an incorporated bar for the attachment of bar bells

f. **Hydrogymnastics** are carried out in therapeutic pools, and to a lesser extent in tanks. This is not to be confused with swimming though swimming motions may be used. The pool should be of good
shallow
should
side. Passive, assistive or resistive exercises can be regulated more accurately if the technician is close to the patient in the pool. The great advantages beyond that of the morale factor is that *movement is possible in all planes with the gravity load lessened* by the buoyancy of the water, and the patient with involvement of the hips and knees may walk in the water.

g **Active exercise with the help of apparatus** frequently offers great advantages. An overhead frame to which a pulley-sling arrangement can be fitted is one of the simplest Frequently used in surgical cases, it could profitably be employed in the arthritic with hip or knee involvement. The familiar pulley weight device of the gymnasium can be used in some ambulatory patients as a form of resistive exercise for the hamstrings or quadriceps. The Kanavel table, shoulder wheel and other simple forms of apparatus may be of great aid in the redevelopment of good joint and muscle function. Scale drawings may be secured from the Council on Physical Medicine and Rehabilitation for the construction of homemade apparatus

Manipulation or mobilization, although not a type of exercise *per se*, may logically be used in some cases following heat, massage, and active exercises. This is to be done gently and with due appreciation for the fine details of anatomy of the particular joint It should rarely be used in the individual with marked demineralization Perfect relaxation on the part of the patient is quite necessary for good results. The joint is "fixed" by the operator's hands or fingers. The periarticular structures are gently stretched, and moved in one or more planes, after which a final thrust is given by the technician. The procedure should not be very uncomfortable, if at all, and no crack or grating is necessary for a good result As practiced by the great English exponents of this art,[78,163] it is of considerable value. As a protective measure for the physician or technician, an x-ray of the joint to be mobilized should be taken. This procedure should be differentiated from the bone cracking of the chiropractor and the adjustments of the osteopath. It does not carry the hazards attached to surgical manipulation under anesthesia.

Legal action frequently follows manipulation of deformed joints. Those individuals employing mechanical apparatus for manipulations on patients with arthritis or deformed joints must realize that this is a very dangerous

Relatively vigorous active exercise is *indicated in fibrositis*. In the more robust patients this should be of the resistive type. Combined with moderately deep kneading and friction massage, the patient can often be kept quite comfortable.

In the subacute and chronic phases of *subdeltoid bursitis or calcified tendinitis*, exercises are of the utmost importance in preventing atrophy of the deltoid and shoulder girdle muscles. Slight to moderate pain is not a contraindication to continued performance. *A pulley-weight system* (attached to a beam in the cellar or to the top of a door jam) may allow almost complete range of motion without too much discomfort. If the patient is allowed to keep his shoulder adducted after the acute phase has subsided, adhesions form and may "freeze" the shoulder.

The patient must realize that physical therapy alone does not cure rheumatoid arthritis or degenerative joint disease, but should be made to understand that periodic, intelligently directed physical medicine will help greatly toward partial or complete rehabilitation.

Medical motion pictures are available from the Film Library of the American Medical Association and from various other organizations. [illegible]

[illegible] Muscle Training," [illegible] " "Occupational [illegible] mentioned are 16 mm silent films. See the more complete listing in the chapter on "Rehabilitation," and at the end of the "Handbook of Physical Medicine and Rehabilitation."

V. MISCELLANEOUS PHYSICAL MEASURES SOMETIMES USED IN ARTHRITIS

A. Heliotherapy.—Ultraviolet irradiaton is an adjunct form of treatment in arthritis. In order to prescribe ultraviolet intelligently, the physician should know something about the spectral characteristics he is using. The commercial market is flooded with many different types for hospital, office, and home use. For his own use and for that of patients in the home he should select or recommend a lamp which has been approved by the Council on Physical Medicine and Rehabilitation.[29] Some of the lamps sold commercially are almost valueless. It is well to remember that ultraviolet is not entirely innocuous since severe reactions can be produced, also that blonds are usually much more sensitive than brunets, and men more than women. The eyes must be protected. Pigment formation and therapeutic benefit are independent phenomena proceeding in the same direction.[27]

Testing for the erythemal reaction is the most practical method for judging the effectiveness of ultravoilet lamps.[15,27,28] In this test a portion of the [illegible]

The so-called sunlamp should require no more than sixty minutes. Sunlamps with an S-1 or R-S bulb have been shown to be satisfactory. The patient should be *warned about possible dangers, and told to use an alarm or interval timer.* Cases of intense sunburn have occurred because the subject fell asleep

Prescription for Ultraviolet
1 The spectral characteristics of the lamp should be known
2. The time is adjusted so as to produce a sub-erythemal, minimal erythemal, or first degree erythemal dose depending on the indication for the condition to be treated
[illegible]
4 The patient's diagnosis is recorded

The chief physiological effect of interest in use of ultraviolet for arthritis is that on the provitamins in the skin, which are converted into vitamin D. Beneficial effects on some metabolic processes occur, and it is thought that anemia may be materially improved [144] There is no substantial evidence that a "tonic" effect may be derived from ultraviolet, but many physicians accept this as clinically proved Albumin is precipitated, and it is for this reason that the eyes must be protected lest cataracts develop Conjunctivitis develops after intense exposure, as in the familiar "snow blindness"

[illegible] often of distinct value to the [illegible] A decrease in pain and muscle spasm and a general tonic effect may follow exposure to direct sunlight, or to other ultraviolet radiation *Some rheumatologists have cautioned against the use of heliotherapy in the acute stage of the disease or in the presence of fever Others have extolled the value of heliotherapy early in rheumatic joint disease* stating that the ultraviolet irradiation of the skin may accelerate the absorption of joint effusion, decrease joint swelling and deformity, and increase joint movement.[80] A maximal erythema dose of ultraviolet radiation has been advised in this connection. The erythema produced by [illegible]

tion with the patient's total regime in either the home or hospital is vitally important[21,33] since undue fatigue must be avoided; *occupational therapy should complement* physical therapy and not be superadded. Two short periods are often preferable to a longer session.

Supervision of Occupational Therapy

[illegible]

5 Use splints for weakened muscle groups where necessary[4,45]
6 Use patient's talents and previous skills
7 Offer generous encouragement for each accomplishment

Crafts Adapted for Specific Joint Mobilization[20,26,44]

1. Shoulders
   - *a* Elevation *knotting, braid weaving, basketry with tall spokes*
   - *b* Flexion knotting, woodworking, printing hand press
   - *c.* Abduction *knotting, weaving, basketry, leather* lacing
   - *d.* Rotation winding wool, wheel weaving
2. Elbows
   - *a.* Flexion. basketry, weaving, knotting, leather working, woodworking, winding yarn
   - *b* Extension hammering, braid weaving, woodwork, planing, printing press, coping saw projects
   - *c* Pronation petit point, crocheting, braiding, screwdriving
   - *d* Supination weave-it-squares, crocheting, needlework
3. Wrists
   - *a* Extension block printing, pottery, wood carving, linoleum block cutting, lathe work, weaving, rug hooking, bookbinding, printing press, sanding, knitting
   - *b* Flexion basketry, carving, braid weaving, bookbinding, hooking
4. Fingers (proximal joints)
   - *a* Flexion paper cutting, puppetry, linoleum blocks, basketry, weaving, knotting, leather lacing, link belts, needlework
   - *b* Extension finger painting, block printing, pottery (coil method), braid weaving, knotting
5. Hips
   - *a* Flexion [illegible]
   - *b* Extension
   - *c* Abduction
   - *d* Adduction
6. Knees
   - *a* Flexion jigsaw, floor loom
   - *b* Extension jigsaw, floor loom, potter's wheel
7. Ankles
   - *a* Flexion and extension sewing machine, foot powered lathe, jig saw, potter's wheel, pianola, motor driven bicycle

the preferable source of ultraviolet light as it also produces heat radiation, which may be beneficial. The *advantages of the carbon arc lamps* are: (1) the full power develops instantly; (2) the lamps are portable and are *not* easily damaged by moving. Their disadvantages are the large amount of current which they consume and the adjustment and replacement of carbons which is necessary from time to time.

The *quartz mercury vapor arcs* are based upon the principle that mercury, when vaporized by intense heat in a quartz container, emits a spectrum which is rich in ultraviolet.[122,177] Because of the ease of handling these lamps and the slight upkeep, they are one of the most widely used sources of ultraviolet light, they require only about one-fourth as much electrical energy as other sources

B. Fever Therapy.—Widely heralded for a few years after the introduction of the hypertherm, the limitations of fever therapy are now well established, and on
Combination with
and antiboitics [116]

at the
rtion of
rheumatoid arthritic patients. Immersion in a hot tub for thirty minutes with the water temperature between 100 and 105° F. will induce a rise in body temperature up to 100 to 102° in thirty minutes, the mild hyperpyrexia can be
from the tub.
ambulatory pat
cases. Other agents for inducing artificial fever include luminous bakers, air conditioned cabinets,[179] electromagnetic induction cabinets, hot packs, electric blankets, and foreign protein injections (typhoid vaccine and pyromen). Fever treatments are usually given in the mornings in institutional practice following a twelve-hour fast or a light breakfast. Exposure to ultraviolet irradiation at the end of the session is thought to offer some advantage to arthritic patients

The physiological and chemical effects of fever therapy have been summarized by Krusen and Elkins.[142] Tachycardia is regularly seen accompanied by hypotension,[13] increased minute volume,[228] and vasodilatation No significant deviations in the blood chemical values for urea, uric acid, creatinine, sugar, calcium, plasma lipids, or serum proteins occur. Considerable decrease in the serum chlorides accompanies the profuse sweating of sustained fever; cool hypotonic saline solution is usually administered throughout the treatment period.[113] Erythrocytes and sedimentation
15,000 or more, develops
ment are not markedly
ly 7 per cent for each
e hours or longer. The
which are permanent.[112]

When the lower extremities are affected and weight bearing or ambulation contraindicated, diversional therapy in bed will prove helpful and pave

ment of the craft, better functional alignment of the working joints is maintained, and postural stress and strain reduced to a minimum[13] Colonial mat weaving and cord knotting are useful starting occupations for this type of patient. Other tasks may be added as the skill and the desire of the patient become known to the therapist. When ambulation is permissible, he is referred to the workshop Its cheerful atmosphere and the stimulus of seeing other handicapped individuals at work will usually prove a strong incentive to effort on his part. Tenseness and neurotic [illegible] The thera- [illegible] movements [illegible] even though motion is carried on against some resistance. The bicycle, jig saw and floor loom are well adapted for hip and knee extension and flexion Resistance may be increased with each as progress occurs. The jigsaw is most useful if fitted with easily adjusted pedals and seat to accommodate individuals of different heights.[8,9] If [illegible] to the pedals so that they may be pulle[illegible] the undesirability of pla[illegible] promote flexion contracture and even subluxation of the knee; her awareness of good bed posture will draw attention to tightly fitting bedclothes, which may induce foot drop and almost always some rotation of the hips

Arthritis of the shoulder often leads to atrophy of the shoulder girdle muscles, a forward droop, some internal rotation, and inability to fully abduct or externally rotate The upper chest may flatten, and the patient is apt to keep his arms adducted all of the time. Weaving, carpentry, tying knots, sanding on an upright board, and the hand printing press encourage good functional use. If necessary, a suspension sling can be used to support the painful extremity so that some activity can be undertaken[8,9]

Large looms, graded for the effort required in their operation, offer exercise for the upper extremities. Loom weaving holds a definite interest for the patient and at the same time may produce graded exercise for prescribed joint and muscle action for arms and shoulders. *For shoulder motion, wheel cushion weaving* is first given on a frame adjusted to the particular limitation and is an easy step to the upright loom which entails more strenuous activity for shoulder flexion and abduction. Any of these crafts are also adaptable as exercise for the flexors or extensors of the [illegible] treatment work for the upper [illegible] led for [illegible] ools to [illegible] houlder and elbow action, as well as hand drills, and the ordinary screw driver which offers pronation or supination of the forearm, depending on the drive of the screw.

When the elbows are involved in arthritis, flexion deformity and some limitation of supination are frequently observed. These deformities are accentuated by the efforts of the patient to pull himself up or to hold on to

There is, however, a marked drop in ventricular filling time [223] In sustained fever a direct lethal or bacteriostatic effect may become effective if the critical temperature of the bacteria is exceeded. Some antibody formation probably occurs, and increased production of cortisone and other adrenal steroids takes place

Contraindications to Fever Therapy[116,205]

- Cardiovascular-renal disease
- Old age (sixty or more years).
- Active tuberculosis.
- Abnormalities of the skin interfering with sweating and abnormal heat sensitivity of the skin.
- Coronary artery heart disease.
- Rapidly progressive late neurosyphilis.
- Marked hypotension
- Aortic aneurysm.
- Liver damage
- Fresh endocardial lesions (danger of embolism).

C. **Low Frequency Currents** —A galvanic or direct current has either a *continuous or an interrupted electrical pattern* It may be produced by a dry cell battery with sufficient voltage to overcome resistance of the skin (a bad conductor) and to stimulate the muscles or nerves beneath; a rectifier is used in the commercial devices to convert alternating house current to direct current. A faradic current differs from galvanic in that it flows intermittently and for very short periods of time. A sinusoidal current is

FARADISM may be used to promote muscular contraction in cases where active muscular movement may be impossible. It is a valuable adjunct to active movements and exercise and aids in the development of a particular muscle group. It may be used in most forms of muscular wasting It must not be substituted for active use of the muscles The Bristow-Smart coil is less painful than the ordinary faradic coil and a surging current is usually employed.[31] It may be used for twenty to thirty minutes with rest periods during the treatment

Faradism stimulates muscles through their motor nerves rather than by direct action on the muscle fibers. In order to stimulate any muscle

stimulated by applying the electrode over a nerve trunk.

GALVANIC CURRENTS stimulate motor nerve fibers only at the time of opening and closure of the current (if the strength of the current remains the same). Galvanism produces contractions in muscle fibers even when the nerve may be degenerated.

tables or chairs. Improperly adjusted canes and crutches may also contribute. Sawing is particularly useful since the extensors of the elbow are brought into play.

Ulnar deviation and subluxation of the small finger joints should be corrected by splints or other cock-up devices (*see* chapter on correction of deformity) Functional alignment of the joints should always be maintained With the fin
sometimes be possible.
the muscles are greatly w
ploy the extensors of the forearm are prescribed. The patient can wear his

is coarse even though splints are worn. Knotting is well-adapted to improve supination

Most arthritic patients are afraid to use painful joints because of the

humerus, flexion of the head, hips and knees, a flat chest and very little, if any, use of the intercostal muscles in breathing Such positions further aggravate the joint phenomena, reduce the circulation and tend to develop flexion contractures and deformities.

Occupational therapy in the arthritic patient must include adequate rest

fully graded exercises will preserve function and aid in restoring muscular power and joint motion During the long period of convalescence, occupational therapy will tend to prevent the development of a neurosis and convince the patient that he is not helpless and that usually he can be

The effect of occupational therapy may be physical, economic, social or mental *Physically*, it is of
ing function to disabled joi
detect skills and capacitie
individuals who may be h
activities of that institution *Socially*, the morale of the patient is improved greatly, as well as that of the other patients in the ward or institution *Mentally*, occupational therapy offers encouragement to the individual, occupying his mind and easing any tendency for emotional stress and restlessness.[9]

Occupational therapy technicians must be properly trained in an accredited school so that they receive training in the fundamental sciences, learn to understand the individual and the physical and mental diseases and the

It is of use in the re-education of groups of small muscles as the interossei or lumbricals of the hand. If the positive pole is applied to the painful joint, one produces vasoconstriction and reduction of nerve irritability. If the negative pole is applied to the joint, counterirritation results from vasodilatation and increase of nerve irritability.[160]

With galvanic currents, treatment is continued for thirty to forty-five minutes using as much current as the patient can comfortably bear; this produces a prolonged cutaneous hyperemia and reflex dilatation of the deeper vessels. It is often used as a form of counterirritation in rheumatoid arthritis

treating marked muscular atrophy in rheumatoid arthritis. The duration of the treatment averages from twenty to thirty minutes several times a week.

D. **High Frequency (Oudin) Treatment.**—This is obtained from the single high voltage (oudin) terminal of a spark gap diathermy apparatus executed through a condenser (vacuum glass) tube. Moving the electrode back and forth in slight contact with the skin produces mild or heavy sparks varying with the strength of the current and the distance of the electrode from the skin.[121] *This acts in a manner of counterirritation and may be* useful in diffuse neuritic pains or subacute or chronic arthritis involving

the treatment of rheumatoid arthritis.[95] In our experience, iontophoresis with mecholyl or histamine results in only temporary subjective improvement similar to that following the use of hot fomentations to the joint. The psychic effect of iontophoresis is great in some instances.

... into the deeper layers of the skin ... ims ... pes of arthritis, but the general consensus of critical opinion is that it has a very limited field of usefulness in the rheumatic state. Less complicated methods *of inducing hyperemia are available.*[111] *One observer noted as good* an effect from injected histamine phosphate (1:1000) as from ion transfer, and superior results to those noted after using an ointment containing histamine.[166]

Neither histamine nor mecholyl should be used in arthritic patients who ... instability, or cardiac ... *but old-fashioned* ... *ions to ion transfer*

a. It is time consuming and difficult to continue after the patient has left the hospital.

b. Other and simpler measures are more effective.

When the lower extremities are affected and weight bearing or ambulation contraindicated, diversional therapy in bed will prove helpful and pave the way for a more active program later. Various supports and equipment for holding work may be improvised or purchased. These support the materials for the patient so that he may devote himself wholly to the development of the craft; better functional alignment of the working joints is maintained, and postural stress and strain reduced to a minimum[23] Colonial mat weaving and cord knotting are useful starting occupations for this type of patient Other tasks may be added as the skill and the desire of the patient become known to the therapist. When ambulation is permissible, he is referred to

the stimulus of seeing other

prove a strong incentive to

introspection disappear as he concentrates at the task in hand The therapist should guide his initial trials to insure that the exact movements prescribed are performed with the greatest economy of effort even though *motion is carried on against some resistance The bicycle, jig saw and floor* loom are well adapted for hip and knee extension and flexion. Resistance may be increased with each as progress occurs. The jigsaw is most useful if fitted with easily adjusted pedals and seat to accommodate individuals of different heights.[8 9] If desirable the feet may be strapped to the pedals so that they may be pulled in reverse The therapist can add emphasis to the undesirability of placing pillows under the knees or thighs, which promote flexion contracture and even subluxation of the knee; her awareness of good bed posture will draw attention to tightly fitting bedclothes, *which may induce foot drop and almost always some rotation of the hips*

Arthritis of the shoulder often leads to atrophy of the shoulder girdle muscles, a forward droop, some internal rotation, and inability to fully abduct or externally rotate The upper chest may flatten, and the patient

... all of the time Weaving carpentry, tying

Large looms, graded for the effort required in their operation, ... *exercise for the upper extremities.* Loom weaving holds a definite interest for the patient and at the same time may produce graded exercise for ... for arms and shoulders *For shoulder*

... to the par-

... ich entails

more strenuous activity for shoulder flexion and abduction. *Any of these* crafts are also adaptable as exercise for the flexors or extensors of the *fingers.*

extremit

action of

be found at a work bench: hand saws, brace and bit, and planes for shoulder and elbow action, as well as hand drills, and the ordinary screw driver which offers pronation or supination of the forearm, *depending on the* drive of the screw.

... and some

c. It places the wrong emphasis on a psychologically impressive procedure.
d. It is primarily symptomatic therapy.
The reader is referred to any of the standard texts for details of treatment and application.[1,18 74 122 135 177]

BIBLIOGRAPHY

1 ABRAMOWITSCH, R. M , and NEOUSSKINE, B . "Treatment by Ion Transfer," New York, Grune and Stratton, 1946
2 ADLER, C · South African Med J , 23, 494, 1949.
3 ALLEN, F. M · in "Physical Medicine in General Practice," ed by A L Watkins, Philadelphia, J. B Lippincott Company, 1946
4. American Academy of Orthopaedic Surgeons, "Orthopaedic Appliances Atlas, Vol 1, Braces, Splints, Shoe Alterations," Ann Arbor, J W. Edwards, 1952
5 ANDERSON, T. P., WAKIM, K G , HERRICK, J F , BENNETT, W. Z , and KRUSEN, F. H.: Arch Phys Med , 32, 71, 1951
6 ANGERER, O. A , BARTH, G , and WINKLER, G : Strahlentherapie, 81, 129 and 82, 461, 1950.
7 BAUMANN, A , and PRESCH, H. R . Strahlentherapie, 81, 143, 1950
8 BAZETT, H. C . J. Am Med. Assn , 111, 1841, 1938.
9 BEARD, G.. Physiother. Rev., 20, 29, 1940.
10 BENNETT, R L . New York J Med , 51, 513, 1951
11. BERGMANN, L : "Ultrasonics and Their Scientific and Technical Applications," translated by H S Hatfield, New York, John Wiley and Sons, Inc , 1938
12 BIERMAN, W. "Medical Applications of Short Wave Current," Baltimore, Williams and Wilkins Co , 1942
13 BIERMAN, W , and FISHBERG, E H J Am Med Assn , 103, 1354, 1934
[illegible] s. Ther , 21, 585, 1940
[illegible] Medicine in General Practice,"
[illegible]
[illegible] Brit. J. Phys Med , 13, 2, 1950
17 BOYNTON, B. L.· Arch Phys Ther , 19, 279, 1938.
18 BROWN, G E , JR , and ALLEN, E. V Am Heart J , 21, 564, 1941
19 BUNDESEN, H N , BAUER, T J , and KENDALL, H W.: J. Am. Med Assn , 123, 816, 1943
20 BUNIM, J S , SOKOLOFF, L., BIEN, E J , WILENS, S L , ZIFF, M , and McEWEN, C. Int. Congress Rheumatic Diseases, New York, 1949
21 BURKART, A S B : "Manipulative Surgery," London, Constable and Co , Ltd , 1932
22 CAREY, E J Am Med Assn , 119, 262, 1942.
23. CHAMBERS, L. A , and FLOSDORF, E W : Proc Soc. Exper Biol and Med , 34, 631, 1936
24 CHERKASKY, M Am J Pub. Health, 39, 163, 1949
25 CLARK, E T , and SWENSON, E. A · "Effects of Heat and Cold on the Circulation," Film Library, Chicago, American Medical Association
26 CLEMMESEN, S Rheumatism, 6, 20, 1950
27. COBLENTZ, W W. in "Handbook of Physical Medicine," 1st ed , Chicago, American Medical Association, 1945
28. ———· in "Handbook of Physical Medicine and Rehabilitation," 1st ed., Philadelphia, The Blakiston Co , 1950.
29 COPEMAN, W. S C.. Brit. J Phys Med , 5, 161, 1942.
30 [illegible]
31. [illegible]
32 [illegible]
33 [illegible]
34 [illegible]
Maryland, W F. Prior Co , Inc , 1940

tables or chairs. Improperly adjusted canes and crutches may also contribute Sawing is particularly useful since the extensors of the elbow are brought into play.

Ulnar deviation and subluxation of the small finger joints should be corrected by splints or other cock-up devices (*see* chapter on correction of deformity) Functional alignment of the joints should always be maintained With the fingers properly supported, simple handiwork may sometimes be possible. Wrist involvement also demands splinting when the muscles are greatly weakened. Exercises and therapeutic work to employ the extensors of the forearm are prescribed. The patient can wear his

is coarse even though splints are worn. Knotting is well-adapted to improve supination

Most arthritic patients are afraid to use painful joints because of the resulting pain on motion. Because of this, the average patient, if left alone, would develop flexion attitudes with flexion of the wrists and fingers, ulnar deviation of the hands, adduction and internal rotation of the humerus, flexion of the head, hips and knees, a flat chest and very little, if any, use of the intercostal muscles in breathing Such positions further aggravate the joint phenomena, reduce the circulation and tend to develop flexion contractures and deformities.

Occupational therapy in the arthritic patient must include adequate rest

fully graded exercises will preserve function and aid in restoring muscular power and joint motion During the long period of convalescence, occupational therapy will tend to prevent the development of a neurosis and convince the patient that he is not helpless and that usually he can be

The effect of occupational therapy may be physical, economic, social or mental Physically, it is of

ing function to disabled jo

detect skills and capacitic

individuals who may be h

activities of that institution *Socially,* the morale of the patient is improved greatly, as well as that of the other patients in the ward or institution *Mentally,* occupational therapy offers encouragement to the individual, occupying his mind and easing any tendency for emotional stress and restlessness.[8]

Occupational therapy technicians must be properly trained in an accredited school so that they receive training in the fundamental sciences, learn to understand the individual and the physical and mental diseases and the

114 Kemp, C R, Paul, W. D, and Hines, H. M: Arch. Phys Med, 29, 12, 1948.
115. Kendall, H. O, Kendall, F. P, and Boynton, D. A: "Posture and Pain," Baltimore, The W
116 Kendell, H. W
Practice," ed 1
117. Kendell, H W
118. King, A J: Br
119. Kolb, S W. J Med. Soc. New Jersey, 41, 406, 1944.
120 Kovacs, R: Med Clin. North America, 21, 1819, 1937
121. ——— Yearbook of Physical Therapy, Chicago, The Yearbook Publishers, Inc., 1940.
122 ———
123 —
124 —
125 —
126 Kr
Charles C Thomas, 1949
127 Krebs, J "Ultraschalltherapie," Osnabruck, Determann, 1950
128 Krusen, F M, Jr, Wakim, K. G, Leden, U M., Martin, G. M., and Elkins,
129
130
131
132 -
133 -
134 -
135 -
136 -
137 -
138 -
139 ——— Proc Royal Soc Med, 43, 641, 1950
140 ———: Arch Phys. Med, 32, 695, 1951.
141 ——— "Physical Medicine and Rehabilitation for the Clinician," Philadelphia, W B Saunders Company, 1951
142 Krusen, F H, and Elkins, E C in "Handbook of Physical Medicine," 1st ed, Chicago, American Medical Association, 1945
143 Lange, K Bull New York Med College, Flower, and 5th Avenue Hospitals, 4, 154, 1943
144. Laurens, H: J. Am. Med. Assn, 111, 2385, 1938.
145 Lautman, M F Arch Phys Ther, 15, 107, 1934
146 Lehmann, J.. Arch. Phys Ther, 3, 57, 1951.
147 Le Quesne, R N, and Granville, M: "Hydrotherapy," 2nd ed, London, Cassell, 1946.
148. Lodeburg, H · Arch Phys Ther., 3, 92, 1951
149 Lowman, C L: J. Bone and Joint Surg, 9, 119, 1927
150 ——— in "Principles and Practice of Physical Therapy," Hagerstown, Maryland, W. F Prior Co, Inc., 1940
151 Ludwig, G D, and Struthers, F. W: Naval M Research Inst. Project, Report No 4, 16 June, 1949
152 Lynn, J. G, and Putnam, T. J: Am. J Path, 20, 637, 1944
153 Martin, G. M.· Med. Clin. North America, 27, 994, 1943.
154. ——— J. Am. Med Assn, 142, 474, 1950
155 ———: in "Physical Medicine and Rehabilitation for the Clinician," ed. by
156 sical Medicine and
157. -
158 Martin, G M, Polley, H. F., and Anderson, T. P.· J. Am Med Assn, 148, 525, 1952.
159. Martin, G. M., Rae, J. W., and Krusen, F. H.: South. M J., 43, 518, 1950.

injuries which they may be called upon to assist in treating. The occupational therapist must also have a broad training in the activities or occupations which may be applied to motivate the patient and must have a knowledge as to which should be selected and changed to control the desired mental processes and reactions of the patient or his specific needs for exercise. She must coördinate all available resources in an effort to hasten the mental, physical and social rehabilitation of the patient; to this end, her imagination and ingenuity will be taxed in adapting processes to carry out the physician's prescription and to aid in the patient's recovery.

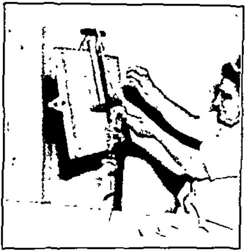


FIG 173 —Occupational therapy in chronic arthritis. The patient should be comfortable and in good posture.

The role of an occupational therapy workshop in general medical and surgical hospitals will depend largely on staff interest. Small hospitals with a bed capacity under 200 usually cannot afford a workshop, and sometimes must compromise by employing a part-time therapist. Poor location of facilities and lack of medical supervision, research, and qualified therapists are serious handicaps.[25,33] If adequate remedial work is accomplished, this form of therapy can be considered curative, and a charge is then justified, psychologically, payment for treatments may enhance the value [illegible]

Recommended changes, in addition to a charge on a cost basis, are: working primarily for endurance, coordination, and manual skill; measurements of improvements by goniometry and score sheets; supplying plastic splints, supports and other aids not available from bracemakers; allowing the patient's choice of craft or activity to prevail, within limits, over the functional objective; and concentration of space to a minimum and of activities to those in which the therapist is most proficient.[31,40] The patient's viewpoint must always be kept in mind by the therapist.[13,31] A background of selected music, transmitted via radio or by means of a record player, may

160. Martucci, A.: Med Clin North America, 24, 1685, 1940.
161 Mead, S : Arch. Phys Med , 33, 5, 1952.
162 Mennell, J. B : "Massage, Its Principles and Practice," Philadelphia, P. Blakiston's Son & Co , 1934

d

169. ———: Arch Phys Med , 31, 687, 1950.
170 Nesbitt, M. E.: Phys Ther Rev , 29, 345, 1948
171 Neyman, C. A.: "Artificial Fever," Springfield, Ill., Charles C Thomas, 1938
172 Nichols, E , Hansson, K. G , and Stainsby, W. J . J Bone and Joint Surg , 16, 69, 1934
173 Nylin, J B . Weekly Roster and Medical Digest (Philadelphia County Medical Society), 32, 1491, 1937.
174 Ollendorf, W. H Proc Soc. Exper., Biol and Med , 72, 432, 1949
175 Osborne, S L. Arch Phys Med , 32, 523, 1951.
176 Osborne, S L., and Frederick, J M. J. Am Med Assn , 137, 1036, 1948
177 Osborne, S L., and Holmquest, H J : "Technic of Electrotherapy and Its Physical and Physiological Basis," Springfield, Ill , Charles C Thomas, 1944
178 Osgood, R. B J Am Med Assn , 95, 992, 1930
179 Ott, V R Brit. J Phys Med , 13, 9, 1950.
180 Palm, M Strahlentherapie, 82, 445, 1950
181 Pemberton, R. Pennsylvania Med J , 44, 1306, 1941
182 ——— in "Handbook of Physical Medicine," Chicago, American Medical Association, 1945
183 Pemberton, R., Coulter, J S , and Mock, H S : J Am Med Assn , 94, 1989, 1930
184 Pemberton, R , and Scull, C W : 'Massage' in "Medical Physics," ed by O

189 ——— in "Physical Medicine and Rehabilitation for the Clinician," ed. by F. H Krusen, Philadelphia, W. B Saunders Company, 1951
190 Polley, H F, and Elkins, E C Arch Phys Med , 32, 146, 1951
...rvous Diseases," 6th ed , London,

J , and Krusen, F. H . Proc.

Arch Phys Med , 33, 73, 1952.
194 Richardson, A W., Duane, T D., and Hines, H M : A M. A Arch Ophth , 45, 382, 1951
195 Robinson, W H Arch Phys Med , 32, 346, 1951
196 Roden, D Brit Med J , 1, 102, 1942
197 Roland, P E. Occupat Ther , 24, 160, 1945
198 Rose, D L., Kendell, H W., and Simpson, W M.: War Med , 1, 470, 1941.
199 Rosenthal, C "Die Massage und ihre wissenschaftliche Begrundung," Berlin, 1910
200 Rudin, L. N , and Cronin, D. J Arch. Phys Med , 32, 729, 1951.
201 Schram, D , and Bennett, R. L.: Arch Phys Med , 32, 227, 1951.

be of considerable aid when dealing with depressed or tense patients.[14 39 43] A manual on the administration of occupational therapy departments is available.[8]

Plastics and other new materials have been added to the armamentarium of the occupational therapist. Recent developments in the field are discussed by various authors.[11,36 42] The use of various procedures in arthritis has been outlined by Snow[41], and others[4,24 37,40] have presented a blueprint for the close inter-relationship of physical medicine and occupational therapy [23 28 35,38 40] Equipment designed to be used for aged persons is described by Haas.[16]

A bibliography of selected books and magazine articles may be secured [illegible]

next chapter.

BIBLIOGRAPHY

1 Barrett, T F Physiother. Rev, 28, 3, 1948
2 Bell, C, and Swaim, L. T: Pub Health Nursing, 32, 243, 1940
3 Bennett, R L Am. J Occupat Therapy, 5, 94, 1951
4 Boershort, L K, and Blau, L Am J Occupat Therapy, 5, 47, 1951
5 Bruggeman, S Occupat Therapy, 18, 305, 1939
6 Bunnell, S Am J Occupat Therapy, 4, 145, 1950
7 Bunnell, S, and Howard, L. D, Jr J Bone and Joint Surg, 32-A, 226, 1950
8 Council on Physical Medicine and Rehabilitation, "Manual of Occupational [illegible]

[illegible] " ed. by W. Bierman and [illegible]

[illegible], 1943

29 Littleton, P. Brit J. Phys Med., 14, 107, 1951.

206. SLOCUMB, C H , and POLLEY, H. F : Med Clin. North America, 28, 309, 1944
207 SMALL, J C Ann. Int. Med , 32, 1037, 1950
208 SMITH, E M.: Arch. Phys Ther , 16, 534, 1935
Soc , 42, 818, 1943.
l , 52, 319, 1952.
ed. by W. Bierman and S Licht, 3rd ed , New York, Paul B. Hoeber, Inc , 1952.
213 SOLOMON, W M , and STECHER, R M : Arch Phys Ther., 21, 339, 1940.
Arch
217 SWEENEY, F X , and STONER, E K · Arch. Phys Med , 32, 206, 1951
218 TAYLOR, H N. 'Physiological Effects of Bed Rest and Immobilization,' in "Physical Medicine and Rehabilitation for the Clinician," ed by F. H Krusen, Philadelphia, W. B Saunders Company, 1951.
219 TREUSCH, J V , and KRUSEN, F. H Arch Int. Med., 72, 231, 1943
220 UDELL, L. "Home Care Program of the Germantown Hospital," personal communication
221 173,
222 1950
223.
224 " ed
225 . H ,
226 Med
227 WAKIM, K G , MARTIN, G M , . H : Arch Phys Med , 30, 135, 1949
228 WARREN, S. L , and WILSON, K M · Am J. Obstet and Gynec , 24, 592, 1932.
229 WATKINS, A. L : Ann. Int Med , 26, 569, 1947.
230 ——— J Am Med Assn , 148, 443, 1952
231 ———: Med Clin North America, 33, 1313, 1949
232 ——— Arch Phys Med , 32, 27, 1951.
233 WEISS, J.: New York State J Med , 51, 506, 1951.
234 WISE, C S., CASTLEMAN, B , and WATKINS, A L : J. Bone and Joint Surg , 31-A, 487, 1949
235 WOLF, H F "The Practice of Physical Medicine," Chicago, Wilcox and Follett Co , 1947.
236. Physiother. Rev , 28, 284, 1948
237.
238. MELTON, C : Proc. Soc
239.
240

30 MacEachern, M T Arch Phys Ther, 20, 753, 1939
31 Mead, S, and Harell, A Arch. Phys Med, 31, 753, 1950
32. Mock, H E, and Abbey, M L in "Handbook of Physical Therapy," 3rd ed, Chicago, American Medical Association, 1939
33. Molander, C O · Arch Phys Med, 31, 757, 1950
34. Neustadt, E · J Health and Phys Educ, 11, 74, 1940
35 Newman, M. K.. Arch. Phys Med, 29, 395, 1948
36 Pattee, G Mod Hosp (No 2), 66, 84, 1946
37. ——— in "Physical Medicine and Rehabilitation for the Clinician," ed by
38
39
40
41
42
43 l Condi-
44 herapy,"
45
46

CHAPTER 22

OCCUPATIONAL THERAPY

By MORRIS A. BOWIE, M.D.

OCCUPATIONAL therapy may be defined as an objective type of treatment prescribed by the physician in an effort to hasten the patient's recovery from injury or disease or to contribute to his adjustment or hospitalization. Occupational therapists are trained to carry out the prescription of the physician by selecting and adapting activities which meet the patient's physical and psychological needs. These activities must be *interesting* enough to induce him to participate actively. While the patient is under observation, the occupational therapist should provide the physician with information as to the patient's reactions and progress.

One of the salutary effects of World War II was to divest occupational therapists from their pre-occupation with arts and crafts. Service institutions with both physical medicine and occupational therapy departments delegated one officer to head both sections. Often these were adjacent to each other so that patients could easily shift back and forth. This seems preferable to separation since coordination of activity is highly important.[1,15,37] Integration of effort on the part of the staff is thus achieved, and both physical and occupational therapists may observe how their particular work is aiding in the rehabilitation of the patient. The growth of community rehabilitation centers will undoubtedly require more integration than in the past, and may necessitate broadening the educational requirements for both types of therapists. Smaller communities may find support of several therapeutic specialists impossible. There is no good reason why physical therapists should not be able to supervise arts and crafts, nor an occupational therapist to administer heat and massage. Therapists of both types should make periodic ward rounds with the orthopedic surgeons and internists to observe and take directions concerning therapy.

Occupational therapy includes any prescribed mental or physical activity which may aid in recovery from a disease or injury and render the convalescence more pleasant. This may be of value at times in keeping the mind of the patient occupied so that he does not constantly think about his illness, in other cases, the work involved results in rehabilitation of the individual. It is important that individuals with chronic forms of arthritis obtain *mental and physical diversion* by means of occupational therapy, the latter, in addition, will be of great assistance in *restoring muscular power* [illegible]

[illegible]

crocheting, knitting, celluloid etching, block printing, chair caning, simple

CHAPTER 23

REHABILITATION OF THE ARTHRITIC PATIENT

By Morris A Bowie, M. D.

The success of the Army Air Forces reconditioning program during World

Ground Forces and the Navy subsequently adopted similar programs. In 1943 Mr Bernard M. Baruch appointed a survey committee "to make a study of the broad field of the use of physical procedures and facilities in the care of the sick, and to make a report on the programs deemed best to advance the development of adequate educational and training plans and places in the United States. Particular attention was to be paid to ways in which physical medicine could contribute most in the care of soldiers and sailors."[4] In April, 1944, Mr Baruch adopted the recommendations of the survey committee and established a central office under the direction of Dr. Frank H Krusen An immediate outgrowth was the formation of a Committee on War and Postwar Physical Rehabilitation and Reconditioning, which began to serve in an advisory capacity to the Surgeons General of the three services as well as to the Veterans Administration The Baruch Committee has acted as a powerful catalyst in stimulating the growth and development of rehabilitation programs throughout the country

woeful lack of facilities for continuing the progress made in the hospital.

in after a busy surgeon amputates a leg and teach the amputee how to

wood work, gardening, pottery, leather work, simple jewelry work, basketry, typewriting, etc. Other simple measures which Hench and Krusen

etc.[15]

Occupational therapy should be prescribed as carefully as physical therapy, using those forms of work or play which will result in the desired exercise for the involved parts *Many patients who will not follow specific directions* concerning ordinary exercises to be prescribed at home, will perform the desired occupational therapy (which often produces the same desired end-result). One must *be careful* that the *patient does not overwork.*

Prescription for Occupational Therapy[8,11,22,23]
1. The type of arthritis should be indicated.
2. Whether the disease is acute, subacute, or chronic.
3 What precautions are to be observed.
4 The type and amount of activity indicated for specified joints.
5 fit into
6.
7

In handling the arthritic patient close teamwork between the internist, orthopedist, physical therapist and occupational therapist yields dividends in returning the patient to normal activity in the least possible time. Ideally, joint rounds are desirable and permit exchange of ideas. Occupational therapy can be employed long before active exercise is indicated, and may contribute greatly in preserving or restoring morale, in the correction of bad bed posture, and, above all, *in reducing the element of fear.*[17,21,29,33,35,45] The field of occupational therapy covers a wide range ... little or no exercise to strenuous work on large

derstanding of the wisdom of graded activity. The *psychology of progressive exercise by means of an interesting occupation* enlists increasing interest and means more than mere exercise.[35,37] Therapeutic tasks adapted to the

ms of self

ic patient

t of work

d mental

nt

a-

exercise what remains of his limb, prevent contractures, teach the use of crutches, and prescribe a proper prothesis. Later he must be instructed in walking, climbing stairs, sitting and rising from chairs, *getting in and out* of vehicles, and all the other varied functions of daily living involving his locomotor system Heat, massage, exercise and electricity are all useful forms of treatment but functional activities have a more basic therapeutic value because they lead to independence. It has been estimated that in the average hospital approximately 10 per cent of all patients require some form of definite physical treatment.

organ disability is much less of a problem than in the mentally ill because of the psychosomatic intricacies. Likewise, the problem of rehabilitation among the orthopedically handicapped is very complicated because of the great variety of conditions which may occur. Physical rehabilitation begins *the day the patient can perform activities without aggravating his disability or disease.* Arthritis and other orthopedic disabilities produce limitations of motion or loss of motion at the joint and weakness or loss of muscle power. The *extent of the limitation and weakness* must be determined and recorded. A goniometer, a tape measure, and chart for indicating muscle strength are needed. A schema for the latter has been published by the National Foundation for Infantile Paralysis.[45]

100% = 5 N = Normal	No apparent deficiency.
75% = 4 G = Good	Complete range of motion against gravity and some resistance. Fatigues more readily.
50% = 3 F = Fair	Complete range of motion against gravity
25% = 2 P = Poor	Complete range of motion with gravity eliminated.
10% = 1 T = Trace	Evidence of slight contractility.
0% = 0 O = Zero	No evidence of contractility.
S or SS = Spasm	Spasm or severe spasm.
C or CC = Contracture	*Contracture or severe contracture.*

... of muscles producing a movement
...eme-

... dis-
ability. Estimations of the degree of joint motion and muscular strength
... in determining exactly what the patient can do. The
... may differ
... ties[10,11] with
an ultimate aim leading to good functional use of the hands and fingers, *self care, and* ambulation and elevation. Teamwork among the physician,
... vocational director,
... cating improvement
...le. A daily activity

inventory is valuable as a means of teaching the patient to meet the physical demands of his daily existence A long narrow sheet of paper with numbered blocks to correspond to each activity is used as a recording silhouette.

A stop watch or chronograph is started at the beginning of each per-

bulletin board with the numbered blocks matching the numbered activities, and is visually available to patient, therapist, physician, and parent, if the patient is a child.

Daily Activity Test[9]
Non-walking Activities
a Bed activities
b Dressing and undressing activities
c Toilet activities
d Eating and drinking activities
e Desk (bedside table) activities
f Wheelchair activities
g Standing activities
Walking Activities
a Progressing activities
b Climbing (stair, bathtub) activities
Elevation Activities
Endurance Activities
Traveling Activities

Hints for a Daily Activity Program[9]
1 Get fundamental equipment[2]
a Chronograph
b Gym mats, 9 x 5 feet. Cover with closely fitting rubber or plastic cover
c Parallel bars, two pairs, adjustable. One should be wide enough to admit a wheelchair
2
3
4
5
exaggerate
6 Repetition of a once-learned activity will establish a habit pattern.
7. Demonstrate frequently and use patients to exhibit their achievements

Rehabilitation in the Hospital.—This portion of the treatment and training schedule is not vocational, but aimed to teach the patient how to meet the needs of daily living. The arthritic patient with swollen, painful knees

London, Cassell and Co, Ltd, 1947.
16 [illegible] Arch Phys Med, 32, [illegible]2, 1951
17 [illegible]
18 [illegible]
19 [illegible] " New York, A S. Barnes Co, 1947
20 [illegible]
21. [illegible]
22 [illegible]
23 [illegible]
24 [illegible]
25 [illegible]
26 Film Library Committee on Medical Motion Pictures, American Medical Association, Chicago
a Council on Physical Medicine and Rehabilitation, "Handbook of Physical Medicine and Rehabilitation," Philadelphia, The Blakiston Company, p 554 1950
27 Greve, B "The Cleveland Rehabilitation Center," in "Rehabilitation of the Handicapped," ed by W H Soden, New York, The Ronald Press Company, 1949
28 Heald, C B. Practitioner, 152, 16, 1944
29 Hellebrandt, F A J A M A, 142, 967, 1950
30 Hill, D F, and Holbrook, W P J A M A, 142, 718, 1950.
31 Houtz, S J, Parrish, A M, and Hellebrandt, F A Physiother Rev, 26, 2[illegible], 1946
32 Institute of Physical Medicine and Rehabilitation, "Self-Help Devices for Rehabilitation," New York
33 Jones, A C J A M A, 144, 994, 1950.
34 Kelly, M M J Australia, 2, 1, 1950
35 Kent, H Brit J Phys Med, 14, 124, 1951
36 Kr[illegible]
37 Kr[illegible]
38 [illegible]
39 Kr[illegible]
40 Krusen, F H, editor "The 1950 Yearbook of Physical Medicine and Rehabilitation," Chicago, T[illegible]
41 Krusen, F H, edito[illegible] tation," Chicago, T[illegible]
42 Krusen, F H, editor [illegible] Philadelphia, W B [illegible]
43 Mead, S Postgrad [illegible]
44 McClellan, W S [illegible]

a hospital bed after his acute process has hospital will utilize definite measures of the previous chapter. Some form of heat (hot fomentations, 4 times daily and subsequently whirlpool or radiant heat in the department of physical medicine) will initiate every treatment period. Muscle setting exercises can be started within a few days unless the process is very acute Meanwhile, passive motion is performed by the nursing staff after the application of heat. Gradually, exercises with gravity partially eliminated are begun, using a smoothed board or large piece of cardboard, which has been powdered If no reaction occurs, light active

in a wheel chair. At first, he may need assistance in transferring from his chair to bed and vice versa and will need help. After a few repetitions he should be able to make this shift without help. When weight bearing is *permissible, a walker is employed With this, some weight can be supported* by the hands An easy forward swing without much bending of the knee or ankle will help to maintain pelvic balance.[12] If crutches seem de-

to the department of physical medicine for physical therapy where the heat and exercise for his trunk and upper extremities can be administered more *efficiently and under supervision. Gentle massage above and below the* knees is initiated after all signs of acute activity have disappeared. The patient tolerates this quite well Swelling disappears fairly rapidly, and painless motion of the knees becomes possible An orthopedist is called in consultation for advice *on the desirability of wearing knee braces. This* may not be deemed necessary for this particular patient, if he exhibits very little flexion deformity on standing Light resistance exercises are started shortly before discharge from the hospital and an increase made in

ing care
to care
in bed,
flexion is the posi-
If necessary,
ed to prevent
further deformity and protect against muscle spasm.

The most practical activity for the patient is chosen as the one to learn first. If the patient has spondylitis of the rheumatoid type and is unable to turn over, but has some use of his hands and arms, a simple wooden cross clamped to the bed will allow him to turn, using one bar of the cross as a pivot. Bed exercises are begun, and when permitted exercises done on a mat in the department of physical medicine. Hydrogymnastics are the

45. National Foundation for Infantile Paralysis, Inc., Publication No 60, New York.
46 NEWMAN, M. K., and MURPHY, A . J. Michigan Med Soc , 50, 1031, 1951
47 OSBORNE, S L.: Rheumatism, 4, 229, 1948.
48 POLLEY, H. F : in "Physical Medicine and Rehabilitation for the Clinician," ed. by F H Krusen, Philadelphia, W. B Saunders Company, 1951, p. 176.
49. POLLEY, H F , and ELKINS, E C.. Arch. Phys. Med , 32, 146, 1951.
50. Physiotherapy Review, Physiotherapy Section· Its Scope and Employment Opportunities. Part II Physical Therapy in a Curative Workshop, Physiother Rev , 27, 23, 1947
51 [illegible]
52 [illegible]
53 [illegible]
54 [illegible]
55 RUSK, H A , and TAYLOR, E. J. "A Directory of Agencies and Organizations Concerned with Rehabilitation and Services for the Handicapped," New York, The New York Times, 1947, Price 10 cents
56. SMITH, O. F G "Rehabilitation, Re-education, and Remedial Exercises," 2nd ed., Baltimore, Wm. Wood and Company, 1949
57. SNOW, W. B : Arch Phys. Med , 26, 220, 1945
58 SODEN, W. H., editor: "Rehabilitation of the Handicapped," New York, The [illegible]
59 [illegible] y W. H Soden, [illegible]
60. [illegible] H Soden, New [illegible]
61 [illegible]
62 [illegible]
63 TREVETHAN, P J in "Rehabilitation of the Handicapped," ed. by W H Soden, New York, The Ronald Press Company, 1949
64 United States Civil Service Commission: Guide for Placement of the Physically [illegible]
65. [illegible] m of [illegible]
66. [illegible] ation [illegible]
67. WARD, F. G Brit J Phys Med., 8, 174, 1945.
68. WATKINS, A L.: New England J. Med , 232, 191, 1945
69. WHITE, B Physiotherapy Rev , 30, 49, 1950
70. WRIGHT, J. Physiotherapy Rev , 25, 22, 1945

ideal type for a patient of this sort because the load of gravity is minimized. If not available, the use of substitute "plastic" covered mats has been suitable in the prevention of friction burns and in the matter of cleanliness since they can be scrubbed daily with soap and water.* A much larger group of exercises can be done on a surface with more width than a bed. A therapeutic pool is especially valuable for the severely ankylosed patient with spondylitis. Supported on a plinth, muscle setting and small muscular movements can be performed, and some passive motion will often be possible. Muscle groups with little or no motion should be massaged while under water. Rope loops, slings, powdered cardboard may be used for the patient while on the mat to reduce the load of gravity. Eventually this patient is ready for *transference to a wheelchair*, if he has fair hip flexion. He may be

one narrow and one wide enough to admit a standard wheelchair, adjustable

sorted chairs, revolving door, turnstile, car body, traffic lights, etc., as additional equipment add interest to the program outlined for the patient.

The average arthritic patient will offer less formidable obstacles to management than those with many ankylosed joints. However, the majority should have the benefit of the stimulating attitude of workshop or rehabilitation center since they will probably follow directions more faith-

Steps in the Physical Rehabilitation of the Arthritic Patient

1. Optimum bed posture

(1) Powdered board, skates, pulley-sling apparatus

b. Ambulation with canes, crutch canes, wheelchair, walker 30 to 60 minutes t.i.d.

daily

CHAPTER 21

SPA THERAPY

Revised by JOSEPH L. HOLLANDER, M. D.

THERE can be no doubt of the value of proper water exercises, well-selected physiotherapy, and a carefully-prescribed routine for the patient with certain types of rheumatic diseases. There is, however, no scientific evidence that the particular mineral content of the water has any specific therapeutic effect. This chapter on spas is included because of its very great historical interest in medicine, and primarily to make a plea for some *basic scientific investigations* in order, once and for all, to clarify the merits, or lack of merit, of the mineral waters

A spa is a health resort where one or more mineral springs are found.[12] The advantages which have been attributed to spas in the treatment of chronic arthritis include:[7,19]

- Rest, the value of a vacation away from home with a complete change of scenery and occupation
- Proper exercise, hydrotherapy and physical therapy.
- Psychotherapeutic value, improved morale of the patient due to his faith that he will obtain a "cure" because others have reported improvement from spa therapy
- Value of mineral baths
- Value of mineral water taken internally (this is probably of very little importance)
- Improvement due to climatic conditions

Unfortunately, American spas are not well known It has been estimated, however, that from 40,000 to 50,000 patients take regular treat- [illegible]

[illegible] can and European spas.[4,11,11]

The value of spa treatment is an established feature of medical management based on ages of experience.

Resort therapy has, during various periods of history, been the central feature of medical care while at other times it has been reduced to the status of a superstition or a social fad or passed into the hands of the charlatan as a panacea

The origin of health resorts probably stems mainly from the priestly healing of pagan peoples The ancient Sanskrit Susruta distinguished six seasons of the year and described their effects upon men and animals[14] *The rainy season from July 15 to September 15 was considered as the most*

An accelerated and more intensive program of physical and rehabilitative measures can be utilized in those patients receiving ACTH, cortisone, or intra-articular injections of hydrocortisone These hormones may reverse the pathological physiology, but do not markedly alter the morbid anatomical changes. Hence, the need for functional rehabilitation and muscular re-education is still paramount. Those patients in whom serious hormonal side effects develop or those in whom incomplete control is impossible due to irreversible articular changes will continue to need supervised rehabilitation.[16, 49] It has been the general experience of many rheumatologists that manipulation under anesthesia is much better tolerated under adjunct hormone therapy; furthermore, the "pick-up" in joint motion obtained by manipulation is usually retained. Manual stretching by the physical therapist may be permissible repeatedly. The reduction in easy fatigability allows the performance of graduated exercises twice or more often daily; progressive resistance exercises may be upgraded much more rapidly.

Home Care of the Arthritic Patient.—Prior to the time of discharge from the hospital a decision should be made relative to the need of the patient for further rehabilitative care such as afforded by the Convalescent Hospital (*see below*). If the patient's score sheet in the "Demands of Daily Living"

of the patient warrants (*See also* p. 482.)

Time Log for the Arthritic Patient at Home

Time	Activity	Time	Activity
7 30	Self Care	3 00	Heat for 20 to 30 minutes *Exercises for 5 to 15 minutes* Occupational therapy
8 00	Breakfast	4 00	Recreation period
9 00	Heat 20 to 30 minutes *Exercises 5 to 15 minutes* Occupational Therapy	6 00	Dinner
10 00	Rest for one hour	7 00	Entertainment
11 00	Activity, reading, writing	8 00	Heat for 20 to 30 minutes *Exercises for 5 to 15 minutes* Occupational therapy *optional*
12 00	Lunch		
1 00	*Siesta for 1 to 2 hours*	9 00	*Self Care; prepare for bed*

Equipment for adequate home care need not be elaborate. A homemade baker, a clamp type lamp or inexpensive infrared bulb will suffice if dry heat is chosen Pails for contrast baths or paraffin dips are usually available A

non-sagging mattress, a foot board, and splints or sandbags to maintain neutral position[3,24,46,61] If indicated, cervical traction is quite feasible

45 National Foundation for Infantile Paralysis, Inc., Publication No. 60, New York.
46 NEWMAN, M. K., and MURPHY, A · J Michigan Med Soc., 50, 1031, 1951
47 OSBORNE, S L Rheumatism, 4, 229, 1948
48 POLLEY, H F in "Physical Medicine and Rehabilitation for the Clinician," ed. by F. H Krusen, Philadelphia, W. B Saunders Company, 1951, p. 176
49. POLLEY, H F, and ELKINS, E. C.. Arch Phys Med, 32, 146, 1951
50. Physiotherapy Review, Physiotherapy Section Its Scope and Employment Opportunities Part II Physical Therapy in a Curative Workshop, Physiother Rev, 27, 23, 1947
51 Report on Rehabilitation by a Special Committee of the British Medical Association Brit Med J, (Supp) 187–201, June, 1946
52 RUSK, H A New York Med (No 6), 3, 15, 1947.
53 ——— Med Addenda Stud, New York Acad of Med, 103, 1947
54 ——— Ann Int Med, 26, 386, 1947.
55 RUSK, H. A, and TAYLOR, E. J "A Directory of Agencies and Organizations Concerned with Rehabilitation and Services for the Handicapped," New York, The New York Times, 1947, Price 10 cents
56. SMITH, O F G "Rehabilitation, Re-education, and Remedial Exercises," 2nd ed, Baltimore, Wm Wood and Company, 1949
57 SNOW, W B Arch Phys Med, 26, 220, 1945.
58 SODEN, W. H, editor. "Rehabilitation of the Handicapped," New York, The [illegible]
59. [illegible] y W. H Soden, [illegible]
60 [illegible] H. Soden, New [illegible]
61 [illegible]
62. [illegible]
63 TREVETHAN, P J in "Rehabilitation of the Handicapped," ed by W H Soden, New York, The Ronald Press Company, 1949
64 United States Civil Service Commission Guide for Placement of the Physically [illegible]
65 [illegible] am of [illegible]
66. [illegible] lation
67. WARD, F G Brit J Phys Med, 8, 174, 1945
68. WATKINS, A L New England J Med, 232, 191, 1945
69. WHITE, B. Physiotherapy Rev., 30, 49, 1950
70. WRIGHT, J Physiotherapy Rev., 25, 22, 1945

with a Sayre or Zimmer head collar, pulley and weights; this may be applied in the sitting position, or in certain cases while recumbent.[43,45] Non-

Baruch Committee recommended that community rehabilitation services and centers be established as expeditiously as possible. The organization should be made up of the following:[4]

1. An administrative staff.
2. Medical services division.
   a. Physical medicine branch
      (1) Physical therapy section
      (2) Occupational therapy section
      (3) Physical education section.
   b. Psychosocial branch
      (1) Clinical psychological section
      (2) Social service section.
3. Vocational services division
   a. Vocational education branch
   b. Special education branch
   c. Vocational guidance and testing branch
   d. Sheltered workshop
   e. Curative workshop
   f. Homebound program.

should appeal to any business man. Many persons on leaving the hospital will eventually be able to return to their former jobs after a suitable period, but some will need to be retrained for new jobs to meet the limitations of their disability. A smaller number can be employed in a sheltered work-

of the 25 activities listed depend upon movements of the joints and strength of the muscles.

Community rehabilitation centers may be of the purely outpatient type or provide dormitory facilities for convalescents recently discharged from hospitals.[33,35] Industrial injuries and compensation cases may comprise in some communities a majority of the problems handled.[35] A comprehensive survey of the work done at the Cleveland Rehabilitation Center, which handles 700 or more patients at any one time, indicates the role a center can play in a large industrial area.[37] Recruitment, screening, and training

CHAPTER 24

SPA THERAPY

Revised by JOSEPH L. HOLLANDER, M. D.

THERE can be no doubt of the value of proper water exercises, well-

great historical interest in medicine, and primarily to make a plea for some *basic scientific investigations* in order, once and for all, to clarify the merits, or lack of merit, of the mineral waters

A spa is a health resort where one or more mineral springs are found [12] The advantages which have been attributed to spas in the treatment of chronic arthritis include:[7,19]

- Rest, the value of a vacation away from home with a complete change of scenery and occupation
- Proper exercise, hydrotherapy and physical therapy.
- Psychotherapeutic value, improved morale of the patient due to his faith that he will obtain a "cure" because others have reported improvement from spa therapy.
- Value of mineral baths
- Value of mineral water taken internally (this is probably of very little importance)
- Improvement due to climatic conditions

can and European spas [6,13,19]

The value of spa treatment is an established feature of medical management based on ages of experience.

Resort therapy has, during various periods of history, been the central feature of medical care while at other times it has been reduced to the status of a superstition or a social fad or passed into the hands of the charlatan as a panacea

The origin of health resorts probably stems mainly from the priestly healing of pagan peoples The ancient Sanskrit Susruta distinguished six seasons of the year and described their effects upon men and animals.[14] *The rainy season from July 15 to September 15 was considered as the most*

of volunteer workers should be a matter of careful selection and well planned orientation.[36]

The interested reader may consult the special reports of the British Ministry of Health,[5] the special committee on rehabilitation of the British

have compiled a directory of agencies and organizations concerned with services to the handicapped.[26] Several new books have been printed in the past years.[15,19 39,40 41,42,56,59]

The Curative Workshop.—Here the patient is recognized as an individual who needs special help. In the case of the patient with arthritis the physical therapist, the occupational therapist, and possibly the social worker and psychiatrist will be mainly responsible for the patients progress. Physical therapy should emphasize muscle training with coordination, relaxation, and walking specially stressed. Special muscle groups may require heavy resistance work.[16] The physical therapist must keep informed of the aims and technics of her co-workers. The occupational therapist conducts a program designed to measure and increase work tolerance. The patient is graded to determine the maximum number of hours he can work within his limitation

Close teamwork among the various therapists is essential. Curative workshops are serving as links between medical-physical rehabilitation and vocational retraining leading to financial self-sufficiency.[6 25 60] State-Federal aid for vocational rehabilitation is available for suitable candidates[13,60] The cost per inpatient in the Vocational Rehabilitation Service

program for a severely

The average stay as in-

and Rehabilitation at

Bellevue Hospital has been 6 to 10 weeks; outpatients are treated about twice as long[35]

A workshop should be a part of the communtiy and not something within it. It should function as an out-patient clinic of non-profit character. Those patients who can afford to pay are asked to do so, but adjustments can be made. It should be a member of the Community Chest. The cost

is borne by insurance companies.[21]

valescent

ot be run

ed equip-

ment is necessary to start, and this can be added to as the needs unfold Basic equipment consists of one or two wooden treatment tables with pads, a tungsten filament lamp of 500 watts, an infrared lamp, assorted weights

strengthening, a Sayre head traction sling, a shoulder

A well qualified

ers with special

skills may help in the beginning to initiate simple occupational therapy. Transportation of patients can be arranged through the Red Cross, local church groups, or individuals.

Audio-Visual Aids in *Physical Medicine*

A.

may

Broa

45. National Foundation for Infantile Paralysis, Inc, Publication No. 60, New York
46. NEWMAN, M. K., and MURPHY, A.: J Michigan Med. Soc, 50, 1031, 1951.
47. OSBORNE, S L Rheumatism, 4, 229, 1948.
48. POLLEY, H F.: in "Physical Medicine and Rehabilitation for the Clinician," ed by F H Krusen, Philadelphia, W. B Saunders Company, 1951, p. 176
49. POLLEY, H F, and ELKINS, E C.: Arch. Phys. Med, 32, 146, 1951.
50. Physiotherapy Review, Physiotherapy Section: Its Scope and Employment Opportunities Part II Physical Therapy in a Curative Workshop, Physiother Rev, 27, 23, 1947.
51. Report on Rehabilitation by a Special Committee of the British Medical Association Brit Med J, (Supp.) 187-201, June, 1946
52. RUSK, H A New York Med (No 6), 3, 15, 1947.
53. ——— Med. Addenda Stud, New York Acad of Med, 103, 1947
54. ——— Ann Int Med., 26, 386, 1947.
55. RUSK, H A., and TAYLOR, E J: "A Directory of Agencies and Organizations Concerned with Rehabilitation and Services for the Handicapped," New York, The New York Times, 1947, Price 10 cents
56. SMITH, O F G: "Rehabilitation, Re-education, and Remedial Exercises," 2nd ed., Baltimore, Wm. Wood and Company, 1949
57. SNOW, W B. Arch Phys Med, 26, 220, 1945
58. SODEN, W. H., editor. "Rehabilitation of the Handicapped," New York, The [illegible]
59. [illegible] y W. H Soden,
60. [illegible] H. Soden, New
61. [illegible]
62. [illegible]
63. [illegible] y W. H. Soden,
64. [illegible] the Physically [illegible] ff.
65. [illegible] ion Program of
66. [illegible] Hospitals
67. WARD, F. G [illegible]
68. WATKINS, A [illegible]
69. WHITE, B. [illegible]
70. WRIGHT, J [illegible]

B. *Motion Pictures*[4, 24, 25a]

	Title	Running Time	Sound or Silent
1	[illegible]	16 min.	sound
2.	[illegible]	21 min	sound
3	[illegible]	22 min.	sound
	[illegible]	22 min	sound
	[illegible]	13 min.	sound
	[illegible]	13 min	sound
	[illegible] ...cal Association)	12 min	silent
	[illegible]	8 min	silent
	[illegible]	4 min	silent
1	[illegible] ...cal Association)	12 min.	silent
11	Underwater Therapy (Committee on Medical Motion Pictures, American Medical Association)	16 min	silent
12	Accent on Use (National Foundation for [illegible]	20 min	sound
	[illegible]	20 min	sound
	[illegible] ...of Canada)	40 min	sound
15	[illegible]	13 min	sound
16	[illegible]	15 min	sound
17.	[illegible]	30 min	sound
18	[illegible]	11 min	sound
19	[illegible]	16 min	sound
20	Reconditioning of the Convalescent (Army Service Forces)	30 min	sound
21	Out of Bed—Into Action (Army Air Forces)	17 min	sound
22	Voyage to Recovery (Bureau of Medicine and Surgery)	30 min	sound
23	Back to Battle (National Film Board of Canada)	23 min	sound
24	Road to Recovery (Army Air Forces)	40 min	sound
25	Hospital School (British Information Services)	11 min	sound
26	Accident Service (British Information Services)	40 min	sound

C *Radio transcriptions* on various subjects in physical medicine have been recorded and are available to physicians, county medical societies, or other qualified groups These have been produced by the Bureau on Med-

CHAPTER 24

SPA THERAPY

Revised by JOSEPH L HOLLANDER, M. D.

THERE can be no doubt of the value of proper water exercises, well-

therapeutic effect. This chapter on spas is included because of its very great historical interest in medicine, and primarily to make a plea for some *basic scientific investigations* in order, once and for all, to clarify the merits, or lack of merit, of the mineral waters

A spa is a health resort where one or more mineral springs are found [12] The advantages which have been attributed to spas in the treatment of chronic arthritis include:[7,19]

Rest, the value of a vacation away from home with a complete change of scenery and occupation.

herapy.

the patient due to his

e others have reported

importance)

Improvement due to climatic conditions

Unfortunately, American spas are not well known It has been estimated, however, that from 40,000 to 50,000 patients take regular treat-

can and European spas [4,12,22]

The value of spa treatment is an established feature of medical management based on ages of experience.

Resort therapy has, during various periods of history, been the central feature of medical care while at other times it has been reduced to the status of a superstition or a social fad or passed into the hands of the charlatan as a panacea

The origin of health resorts probably stems mainly from the priestly healing of pagan peoples The ancient Sanskrit Susruta distinguished six seasons of the year and described their effects upon men and animals.[14] *The rainy season from July 15 to September 15 was considered as the most*

ical Education of the American Medical Association, and may be secured without charge on a loan basis. The running time is approximately fifteen minutes. These are suitable for local broadcasting under the sponsorship of the local medical society ...

de

home Such small elevators can be made large enough to accommodate a

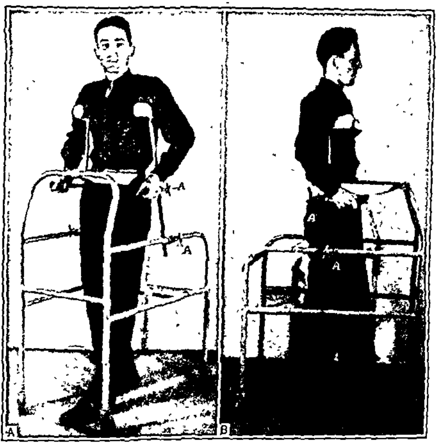


FIG. 174.—A metal walker which is very useful in reëducating the arthritic patient to walk after he has spent some time in bed Arm and hand rests are adjustable by screws at *A* Canvas strap holds patient and gives him added confidence Metal seat behind patient may be used if patient tires

wheel chair. The escalator can be made so that the patient may ascend while seated in a chair These services are available in practically all large towns.

Counterbalanced Stairway Chair.—One of the misfortunes in arthritic patients with involvement of the knees or hips is that they cannot climb steps; thus the patient is often confined to one floor. This can be overcome rather easily in the average home by building an apparatus by means of which the patient can be safely transported up and down a stairway. About

dangerous to health and the change to a dry climate was strongly recommended. The laws required that the king sojourn in such a dry locality during this season and apply suitable measures of hygiene. There was, of course, a considerable migration following the king. *Representatives of the healing art flocked to these places since bathing, massage and general care of the body were emphasized*; these localities thus developed into health-resorts.

In most other instances, the origin and growth of health resorts were probably religious, with therapy centering about the sacred spring or well. The

springs and wells attained special significance through such mysterious and unusual properties[14] Temples were built for the priests and quarters were provided for the multitude of pilgrims who made visits to the healing waters. At that time, emphasis was laid upon the divine influence emanating from the waters or some supernatural elements so that the individual was oblivious to the beneficial effect of the rest, lack of responsibilities of

and health resorts.

As Christianity spread, these pagan healing waters became Christian healing waters and the healing effects were often attributed to some patron saint The physical ministrations were now minimized or even abolished in some cases and mere visit and ceremony at the spring or well were

physicians. Following the works of Paracelsus, the mineral content of medicinal waters was stressed, but in the eighteenth century, many famous waters were found to contain no more mineral ingredients than in some city water supplies

Even in ancient times, spa therapy included the use of mineral waters internally and their application externally in the form of various baths at a great range of temperatures The virtues of mineral waters containing copper, silver, gold, sulfur, etc., were commented upon by Hippocrates, Aristotle, Strabo, Theopompus, etc., and Galen eulogized certain of them

by the greatest architects and engineers. The public baths were then often great clubs with lounges and libraries.

Later, the vapor bath with plunge, massage and rest became very important in daily life in the Mohammedan countries.

One of the earliest medical treatises on baths was written in 1489 by Savonarola of Padua, this dealt mainly with the thermal waters of Italy.[5] Charlemagne temporarily revived interest in the French mineral waters by having a bath constructed for his family at Aix la Chapelle.

half the width of the average stairway is usually required for this purpose.

The apparatus is made as follows: Two long two by fours are fastened parallel on to the steps, beginning at the top, and extending to the bottom of the stairway; they are placed about 10 inches apart. A wooden chair is made with two short legs posteriorly and 2 long legs in front. Roller skate wheels are fitted into the bottom of each leg so that the chair will slide up or down on the two by fours on its roller skate wheels. A wooden guide is placed along the outside of the two by fours so that the chair cannot slip off the track.

At the top of the stairs is placed a grooved wooden pulley wheel. Passing over this is a ¼ inch marine tiller line (non-stretching cable) which is fastened at the upper end to the chair and at the bottom of the stairs to a small wooden box whose width is equal to the distance between the two by fours (about 10 inches). The box has attached at its sides (below) roller skate wheels. The box is filled with steel plates sufficient in amount to counterbalance the weight of the patient who will be placed in the chair at the top. The rollers on both sides of the bottom of the box slide along the inner side of the track made by the two by fours.

[illegible]

extremely important as the main hazard of the apparatus is that someone may unhook the empty chair at the bottom (allowing the weight box to crash rapidly to the bottom).

Inasmuch as the patient is counterbalanced by the weights in the wooden box, the individual is slowly taken downstairs in the chair as the counterbalanced box rises. When the individual is ready to come upstairs again, [illegible]

the patient has limited hip and knee motion to 45° or less. The ordinary stair riser is 8 inches. Even with limitation, the patient can usually lift the leg 3 to 4 inches, by raising on the toes and lifting the pelvis. A block of wood, four inches thick and a foot long is attached to an upright handle 3 to 4 feet long. This the patient uses to get from one step to the next, and then lifts the block to the next step to repeat the process.

[illegible]

confined to bed. The essential features are, a firm bed which can be raised and lowered at either end, a Bradford frame with handles or a bar for lifting.

Other apparatus which may be useful in the home, clinic or hospital where large numbers of arthritic patients are seen include:

The High Seat Chair.—In such a chair, the legs are especially long (the seat averaging 25 inches from the floor). The primary purpose of this chair is comfort for individuals with ankylosis or deformity of the knees or hips. These patients can sit on high chairs with relative comfort without bending the affected joints, also the patient can rise from such chairs without as-

Most European spas are under some form of governmental control which has maintained a high scientific interest and has aided in preventing their abuse; under this system, there is provided adequate means for the care of the poor and those of moderate means.

resorts in this country, including Hot Springs, Virginia, Arrowhead Springs, California; Mineral Wells and Marlin, Texas; French Lick, Indiana, Glenn Springs and Sharon Springs, New York; and many others. The *Thermal Water Reservation at Hot Springs, Arkansas, is owned by the government The Spa at Saratoga Springs, New York, is owned by the state*, while the *state of Wyoming controls the springs at Thermopolis, Wyoming Excelsior Springs, Missouri, is a municipal development* The watering place at Mount Clemens consists of a group of hotels or sanatoria which have grown up around the source of mineral waters in that area [11]

The Federal government owns the mineral water and lands at Hot Springs, Arkansas, this water is metered to a government owned charity bath house and to many hotels and bath houses under private ownership. These latter are operated for private gain but are under regulations of the U S Department of the Interior The springs and lands at Saratoga Spa are owned by New York State.

During World War II, some of the larger spas in the United States were

It is not the purpose of this discussion to recommend any certain spas.

Jarman has defined a spa as an institution which is built around a mineral spring or springs, equipped and staffed as to utilize the water from these springs together with other therapeutic agencies for health purposes.[16] Although the springs determine the location of the spas, it requires more

most spas, emphasis is placed upon the triad of rest, exercise and diet [17]

sistance. Such chairs can easily be made by fastening a block of the required height under each leg (usually 3 or 4 inches).

The Metal Roller Chair.—Bathing the arthritic patient is often difficult. This is simplified by lifting the patient on to a metal chair with rubber rollers inserted into its legs so that the chair can be wheeled to the bathroom and into a stall shower. Following his shower, the patient can be dried or wheeled back to his bed where final drying may be completed. This is

to allow the patient support while bathing

The High Toilet with Arm Rests.—The toilet is raised (built up) so that the seat is about 10 to 12 inches above the usual position. The arthritic patient with involvement of the hips or knees can sit comfortably upon this if horizontal bars (metal) are placed as rails (just below shoulder level) which the patient may grasp in sitting or rising.

The Individual Pool.—A small pool lined with stainless steel can be built inexpensively by any large institution. With the aid of a plumber and engineer (usually available in hospitals) such a small pool may be built easily. The water is maintained at any desired temperature (usually about 100 degrees for exercises) and the patient is lifted into and out of the pool by a Bradford frame so that no discomfort is caused by handling. If chlorine is added to the water, it may be used all day in the clinic.

A portable, waterproof canvas tank, 24 inches deep, 6 feet long, and 4 feet wide, suspended from a frame 43 inches high, has been devised.

folded. The bottom of the tank is kept rigid by folding boards held under it by a folding steel framework. An angulated Bradford frame is suspended from one edge of the frame and rests on the bottom of the tank. The patient's head rests upon canvas straps which are slung across the head of the tank. Water may be run in by a garden hose. The tank may be sterilized by chlorinated lime, and washed with soap and water.

Combination Reading Lamp and Book Holder.—Arthritic patients who are confined to bed and deprived of free use of the hands will appreciate such a device which is built on a floor stand. The patient can read a magazine or heavy book, while in any position, without holding the book. Special lamps ... be attached ... now available ... flat in bed can look straight up, and through the glasses read a book supported on his chest.

BIBLIOGRAPHY

1. ALPERS, P. M. J. Australia, 1, 286, 1951.
2. AREY, M. S.: "Improved Equipment for the Physically Handicapped," National ... -k.

"Home Care in Rheumatoid Arthritis," Annual Report, New York, 1947

Varying with their location, spas utilize various components such as sunshine, temperature, relative humidity, sea bathing, mineral springs, etc. *One of the most important secrets back of the success obtained at spas is an abundance of rest.*[23]

Mineral waters have been given in several ways: (1) internally; (2) baths; (3) inhalation; (4) hot packs; and (5) as gargles and colonic or vaginal irrigations. Drinking of the mineral waters in varying amounts is their most widespread use.

Some individuals believe that certain natural waters have properties of therapeutic value, although this has not yet been proven scientifically. Others are firmly convinced that the most important chemical in these mineral waters is water itself and that there is a great psychologic effect produced by prescribing the drinking of mineral waters according to exact instructions.

The drinking of water is almost dramatized at some spas, being done according to definite directions as to amount and time, and in some cases, to the accompaniment of music. Water is one of the best diuretics, and some waters are definite saline cathartics.

Reynolds has defined a health resort as a hospital or spa with natural therapeutic facilities for the care of chronic diseases which offer convenient, attractive and healthful accommodations in an environment which would attract individuals requiring rest and rehabilitation and those searching for athletics, sports or even social advantages.[23]

The usual program in most health resorts is that the patient selects a regularly practicing physician of the vicinity experienced in the program of treatments offered by the spa. *Very few of these health resorts have provision for charity patients.* At *Hot Springs, Arkansas* and at the *Saratoga Spa* in *New York, the charity patient must submit proof of his inability to meet cost of therapy, and medical attention is provided without charge by staff physicians.* **Living accommodations during this treatment period must be provided by the patient or his friends or by charitable organizations.**

At most spas, the majority of individuals must seek living accommodations in nearby hotels and boarding houses. Very few spas are professionally staffed; most rely in great part upon the local profession for the care of their patients.

The seasonal character of most spas aggravates their financial uncertainty inasmuch as many of these are open for less than half of the year with patronage of the recreational features naturally being much higher in the vacation months.

It has been estimated that in 1930, 100,000 Americans were treated at European spas, spending an average of $1000 each.[3]

The economic set-up of most spas is such that they cannot care for the indigent and low income classes without outside subsidies or changes in their present type of operation; *there are some provisions for low income classes and the indigent at two spas in this country* (*Saratoga Springs*, New York, and *Hot Springs*, Arkansas). In Europe, insurance and relief organizations coöperated with the spas by extensive arrangements so as to provide treatment for convalescents [illegible] ill among the low income brackets. As a basis of the [illegible] Springs Authority by the [illegible] C[illegible] ial arrangements were provided for [illegible]

Ant and McClellan have stressed the importance of *adequate physical equipment and their intelligent use* in the successful administration of treatments in a health resort[1] They have outlined the *requirements for such a spa*, including provisions for mineral waters, packs, massage, hydrotherapy, baths, inhalations, mechanotherapy, drinking halls, swimming pools, etc.

Baudisch has attempted to arouse interest in the *importance of the trace elements in biologic activity*, with special reference to the use of natural mineral water to supply these elements.[2] He has compared the effect of vitamins, which are present in our bodies in only minute amounts, with the possible effects of various minerals which are present only in small amounts in the human body, he believes that the importance of these trace elements is as yet undetermined. He has called these mineral elements "inorganic vitamins" and is firmly convinced that their insufficiency may be of much greater importance in the human economy than is at present recognized

Mineral wa[illegible]
thermal wate[illegible]
this country [illegible]
calcium, magnesium or sodium

Thermal springs are those with temperature above 70° F.; hot springs indicate those with temperatures above 98° F.

Mineral waters differ in their color, taste, temperature, odor, etc

Gases are dissolved in many mineral waters; those considered of most importance therapeutically include carbon dioxide, hydrogen sulfide and radon (radium emanations)

The term "chalybeats" was used in former years to refer to waters with at least 10 mg of ferrous or ferric iron per liter

Strong brines may assist in the venous return as well as aid the lymphatic circulation by pressure on the lower extremities

Some mineral waters exert a laxative effect and it has been claimed that they may influence gastric motility and the circulation Several thousand years of empiric observation is in back of their use for rheumatic disorders so that despite the lack of any scientific proof of their effectiveness, the use of mineral waters in rheumatic diseases may have more rational value than the mere effect of a warm bath and relaxation, although this is yet to be shown decisively

Underwater therapy is a feature of the treatment at most spas. This includes hydrotherapy, therapeutic exercise and massage. Some individuals have defined hydrotherapy as the outward application of water to the body; **balneotherapy** is used to indicate bathing at a resort or a spa.[35]

[illegible]

tan, Vichy; and Aix.

Hydrotherapy is one of the most useful forms of physical therapy in the treatment of rheumatic diseases. It is unfortunate that so few hospitals are equipped with facilities for such therapy[15 24 34]

stood, psychogenic rheumatism will serve as an useful term, at least until a better one appears.

Psychogenic rheumatism may be defined as the musculoskeletal expression of functional nervous disorders, tension states or psychoneuroses It implies that such symptoms as pain, stiffness, subjective sense of swelling or limitation of motion in muscles or joints may be caused, intensified or perpetuated by mental influences It does not mean that an organic joint or muscle disease has resulted from mental conflicts [4] The term should be restricted for those cases in which the actual disability results from functional symptoms, it should not include minor nervous reactions which develop in patients with chronic arthritis

Incidence —Psychogenic rheumatism is one of the common causes of rheumatic complaints along with osteoarthritis, rheumatoid arthritis, rheumatic fever and primary fibrositis In a recent study of 500 consecutive civilian patients with rheumatic disease the diagnosis of psychogenic rheumatism was made in 13.4 per cent of cases, while osteoarthritis was diagnosed in 25 per cent, rheumatoid arthritis in 20 4 per cent and primary fibrositis in 18 2 per cent.[1] The incidence was high among soldiers admitted to military general hospitals for musculoskeletal complaints Psychogenic rheumatism was the most frequent cause of disability (34 per cent) in 450 soldiers with rheumatic complaints admitted to one army general hospital during World War II [2] Previous to admission all had received organic diagnoses, usually arthritis The incidence of functional symptoms displayed by the muscles and joints was comparable to those demonstrated by other systems, including the gastro-intestinal and cardiovascular. In an army hospital located overseas, Short found that 16 5 per cent of soldiers with articular complaints were incapacitated because of psychogenic rheumatism, an incidence equal to that for osteoarthritis and exceeded only by that for rheumatoid arthritis.[25] Flind and Barber found the disability to be psychogenic in 42 per cent of patients admitted to a Royal Air Force special rheumatic center [9] At the United States Army's Rheumatism Center during World War II approximately 1 out of every 7 soldiers was disabled because of psychogenic rheumatism At one center it was the second most common diagnosis, being exceeded only by rheumatoid arthritis, at another it was the third most common diagnosis, being exceeded by rheumatoid arthritis and osteoarthritis [19]

Psychopathology.—There are three requisites for the development of any psychosomatic disorder: (1) A psychoneurotic predisposition, (2) an exciting emotional conflict, and (3) restriction of outward expression of the conflict. Regardless of whether they are normal or psychoneurotic, all persons seek flight from the mental sufferings occasioned by disturbing life problems If emotional tension is dissipated by outward expression, muscular or verbal, all is well: A person feels better by taking some form of action or doing something about it, thereby "getting it off his chest" However, if there is constant restriction of emotions, the tension gradually mounts and, in a psychoneurotic type of individual especially, the eventual outlet takes form in somatic complaints When bodily symptoms emerge, the psychoneurotic individual tends to focus his attention on them and thereby obtains some degree of relief from his inner mental tension. Not fully understood, but undoubtedly complex, are the psychophysiologic mechanisms by which psychic disturbances are displaced into physical symptoms. Re-

The warm pool relieves muscle pain and spasm in arthritis and eliminates most of the body weight by the buoyancy of the water.[27,30] In subacute and chronic rheumatoid arthritis, the increased arc of painless movements possible in under-water therapy aids in hastening the absorption of joint exudates, reduces muscular atrophy and helps to prevent adhesions.

Most spas utilize manual massage by trained masseurs as an important part of their program. The machines devised by Dr. Gustaf Zander of Stockholm for active and passive exercises of voluntary muscles are used very little at present because well-trained masseurs can accomplish their feats more safely, less expensively and more effectively.

Jarman believes that the spa is not the best place to treat the patient whose disease is so active as to require splints.[16]

Singer and Phillips have summarized their concept of Thalassotherapy *(the utilization of ocean climate in treating or preventing disease).* Histori-

sanatoria prior to the war. *In Belgium alone, the forty miles of seashore had more than 40 sanatoria with 3500 beds.* In contrast to this, *there are*

Two types c ... ative or protec-
tive climate ar ... southern Cali-
fornia and of ... *type,* as is the
summer on any seashore in this country. A winter stay at seashores other than Florida and southern California is considered to be stimulating.

Thalassotherapy offers sun, air, surf bathing and outdoor exercise, baths in heated sea water and mud packs and baths. There are practically no known contraindications to a stay at southern seashores in any season or at seashores in temperate regions in the summer and early fall. If any individual with rheumatic disease, and particularly fibrositis, is continually made worse by a stay at the seashore, it is common sense for that individual to avoid this region. *Rheumatic patients are seldom made persistently worse by a stay at the seashore during the summer months,* despite the popular concept among patients in this country that those with rheumatic disease must avoid the shore.

The *main climatic factors at the shore* are the water, sun and air. Sea air usually is relatively free from pollen, allergens, dust and gaseous

between inland and ocean surface temperature is an important feature of the seashore; in the summer, this reduces the daily fluctuation of temperature, moderating the mid-day heat. *The sea breeze has been likened to a*

cently it has been suggested that *emotions may affect the autonomic nervous system*, either directly or *through the medium of the endocrine system*, and thereby produce increased muscle tension, vasospasm, or metabolic changes *in various organs and structures.* (See also discussion of Adaptation Syndrome in chapter on Endocrine Glands in Rheumatic Diseases, p. 522.)

"Smoldering resentment" is regarded by Weiss as the common basic

He pictures resentment as

or one reason or another the

this creates a continuous

sense of hostility which prevents the patient from relaxing. A state of constant muscle tension is created which leads to symptoms of aching, tenseness, stiffness and muscular fatigue. Although the underlying psychologic mechanisms are open to question, the somatic effect of chronic muscle spasm appears probable. *Most physicians themselves have experienced muscular aching and fatigue in one region or another following periods of prolonged emotional tension.*

Diagnosis and Clinical Picture.—Diagnosis depends on (1) the absence of organic disease or insufficient disease to account for the disability, (2) the functional characteristics of the presenting complaints, and (3) the positive diagnosis of psychopathology The mere exclusion of objective evidence of organic disease is not enough.

Absence of Organic Disease or Insufficient Diseases to Account for the Disability.—It is convenient to divide psychogenic rheumatism into 3 subtypes: (1) Pure—when there is a total lack of clinical, laboratory and roentgenographic evidence of organic joint or muscle disease; (2) Superimposed—when incapacitating psychogenic symptoms are associated with a nondis-

tional prolongation) Among 1021 cases of psychogenic rheumatism observed at an army rheumatism center during World War II, the relative frequency of these subtypes was as follows. Pure 41.6 per cent; superim-

osteoarthritis, diseases which of themselves usually have only nuisance value, are more commonly associated with superimposed disabling func-

the lumbar spine, for example, may be accompanied by persistent and marked disability far out of proportion to that usually seen with such changes, especially if the patient is aware of the roentgenographic changes and develops an anxiety reaction to them. Thus, an emotionally unstable

natural air conditioning mechanism at the shore, maintaining a convenient degree of humidity, purifying the air and producing a desirable temperature range

Although an area up to 50 miles or more inland may be influenced (as regards weather changes) by the vicinity of the ocean, the cooling effect of sea breezes can usually be felt up to 4 to 6 miles inland

The reflection of radiation from the beach sand and from the surface of the water results in a distinct difference in the *quality of the sunshine on seashores* in comparison with sunshine elsewhere

Although sea water contains from 2 to 4 per cent salt (mainly sodium and potassium chloride), *there is no absorption of the mineral content even following prolonged bathing* The effects of sea bathing have been attributed to other factors, including the temperature of the water and the difference between the skin and water temperature, counterirritation (from

Old medico-religious ceremonies (Persian, Assyrian and Babylonian) depicted the use of *moors* and *muds*, while the Romans employed *heated sea mud* extensively in the extravagant bath houses of that city (150 A D.). In 1935, over 300 European spas were applying *pelotherapy*,[21] particularly in Czechoslovakia, Germany, France, Italy, England, Norway, Sweden, Hungary, Russia and Rumania. Less than ten years ago, the Austrian Government registered Austrian muds and selected these as state reservations

The term *peloid* is used to designate all types of peats, moors and muds which are employed for therapeutic purposes *Peloids* are substances

Mud is composed mainly of sand, clay and kaolin *Peats* and *moors* are made up of mosses, algæ and diatomaceæ with the débris of vascular plants *Sea muds* show a mixture of botanical and mineral contents Muds are neutral in reaction while liman is alkaline and peats and moors are acid

Fango is obtained from the beds of mineral springs of volcanic origin in the Rocky Mountains or in Italy and consists of a fine, homogeneous and nearly pulverized mud *Liman or sea mud* is mainly mineral and is deposited on the bottom of inlets and shallow bays, it contains microscopic shells of cadavers of the one cell life of the ocean, mixed with sand, quartz and sea vegetation detritus. Liman has been obtained in Sweden, Russia and in this country in New York and New Jersey *Peats or moors* are mainly vegetable muds and result from the moldering of marsh vegetation, they are characterized by their carbonized content of plant particles of bogs (and is obtained from Florida, southern California and Germany)

person may elevate a minor organic discomfort to the status of a major disability. The experienced physician is able to detect incongruities betw

cu

sidence of a rheumatic disease such as rheumatoid arthritis, rheumatic fever, gout, etc. Following some minor injury, the symptoms of which ordinarily would subside within a few days, the patient may have intractable symptoms for months or even years. Functional prolongation of disability following minor back injuries are particularly common, recovery sometimes fails to result even after prolonged bed rest or immobilization Characteristically, a gradual change in the quality of the symptoms develops as the psychogenic factors usurp command of the disability The initial symptoms may be typical of organic disease, but eventually the complaints change their colors and assume obvious functional characteristics

Qualitative Functional Characteristics of the Disability.—The complaints

that he has pain, but on close questioning the pain may have functional characteristics and is unlike that which occurs with rheumatic disease Such sensations as weakness, fatigue, pricking feeling, tingling, numbness, deadness, burning, fullness and pressure may be interpreted as pain by the patient. After analyzing the symptoms it may be obvious that the patient complains of paresthesias rather than pain It may be constant, "bad all the time," "present day and night," or it may be fleeting, lasting for only a few seconds at a time. Local heat and various physiotherapeutic measures

joints.

When aching and stiffness are the predominant symptoms, they usually fail to fluctuate with external environmental factors such as weather

Varying degrees of muscle spasm may be found on physical examination and when marked may be accompanied by limitation of joint motion The patient's attitude often is tense, anxious, defensive or antagonistic. He may display fear or resistance to examination He may cry out with pain on palpation or movement of a joint which appears normal, or he may overreact, jump, grimace or groan on slight palpation of some muscle group, overdramatization and lack of consistency of painful motions are characteristic. He may protest to being hurt by grasping the examiner's hand, a

One of the best detailed descriptions of mineral waters, peloids, and climate is presented by Vogt.

The *biologic effects which have been claimed for muds are due mainly to their physical properties:*

1. Because of their specific heat which is much lower than that of water, a *60° C temperature can be well borne in hot mud.*
2. *Mud packs grow cool very slowly* because of their poor heat conductivity.
3. Mechanical stimulation of the contained microscopic shells, spicules and quartz grains acts as a counterirritant.
4. It may be applied to practically any part of the body because of its *plasticity*

European health resorts used mud baths extensively. A mud bath or a mud pack will cover several joints at once and can be applied at a higher temperature than water. Rectal temperatures of 103° F. can be obtained easily within thirty minutes by a mud pool

European resorts employ more mud therapy in arthritis than American resorts. Krusen feels *that there are no particular advantages of mud packs over cleaner and simpler methods of local application of heat.*[18]

There is no convincing proof as yet that any of the constituents of mud particularly enhance its effectiveness.

Neuwirth[23] believes that the general reaction produced by a warm mud pack is comparable to a foreign protein reaction. The increase in body temperature during the mud pack results in a stimulation of the general circulation, with increased metabolism

It is claimed that the application of heated mud results in an increase in body temperature, and a lowered blood pressure.

Singer lists the *contraindications to pelotherapy* as follows:[34]

1. Acute diseases or subacute stages of chronic diseases.
2. Marked physical debility or early convalescence
3. Marked peripheral vascular disorders
4. Cardiac decompensation.
5. Pregnancy
6. Advanced tuberculosis

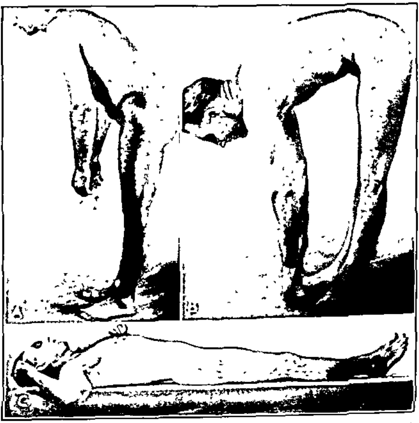
Because the specific heat of mud is much less than that of water, a temperature as high as 50° C. can be borne in hot mud while a temperature of 44° C. is about the maximum borne in hot water.[33] During their protracted application, mud packs grow cool slowly because of their poor heat conductivity The temperature drops only a few degrees in several hours. The number of calories transmitted per unit time is less with mud than with water, and it has been stated that the "heat of mud does not invade the skin vehemently, but sneaks in."[33] Thus, by use of a mud pack, one may obtain conductive heat which promotes absorption of exudates, and relieves local pain as a warm wet compress, being more soothing, penetrating and relaxing than dry baking.

BIBLIOGRAPHY

1. A[illegible] Am. Med. Assn. 123 605 1943
2. B[illegible] ...rt of the
3. B[illegible] Y., 1930

"touch-me-not" reaction. In a minor percentage of cases major hysterical conversions develop (especially in compensation and military cases); these are manifested by bizarre limps, gaits or postures and hysterical contractures. (Figs. 175 to 178.)

Positive Diagnosis of Psychopathology.—Evidence for the diagnosis of psychoneurosis must be established. The functional nature of the complaints (as already discussed), the existence of associated psychoneurotic manifestations referable to other systems, a history of emotional instability in the patient's past history and family history, the presence of an exciting emotional conflict, etc., allow this to be accomplished.



[illegible] to
an
ion

4 BEHREND, H. J : Arch Phys. Ther., 17, 501, 1936
5 BELL, J : On Baths and Mineral Waters, Philadelphia, W. H Porter, 1831
6 British Health Resorts, Spa, Seaside, Inland, ed by R F Fox, London, J and A Churchill, Ltd., 1937.
7. BUCKLEY, C W.: Jour State Med , 44, 508, 1936
8 COLLINS, W D Mineral Waters in 1923, U S Geological Survey, Department of the Interior, p. 109
9 Council on Physical Therapy, Jour Am Med Assn , 113, 40, 1939

27 RAY, M B Lancet, 1, 683, 1940

York, pub-

1944
1944
d 2, Berlin.

37 VRTIAK, E , BENSON, S , KOBAK, D , and CARLSON, A J Arch Phys Ther , 19, 325, 367, 1938
38 WALLACE, A W Jour Am Med Assn , 106, 419, 1936

Psychoneurotic Predisposition.—Previous neurotic traits can be elicited



FIG 176



FIG 177

FIG 176 —Another example of hysterical bent back

FIG 177 —Hysterical bent knee The patient could straighten the knee fully while lying but not while standing He had a bizarre gait, semi-squatting with each step (Boland, courtesy of Ann Rheum Dis)

or hysterical episodes prior to the onset of psychogenic rheumatism; these ranged in severity from attempted suicide to interruptions in schooling be-

with psychogenic musculoskeletal He believes that they possess obsessional trends with self-limitation and restriction of feeling During

childhood they tend to be shy and later in life are overorderly, punctual and clean, and seek a quiet type of existence. He finds this personality to be in contrast with that exhibited by patients with functional dyspepsia or peptic ulcer; these are described as showing compulsive drives, overemphasized activity, efficiency and independence, being especially susceptible to threats against security and to changes of authority. But certainly not all

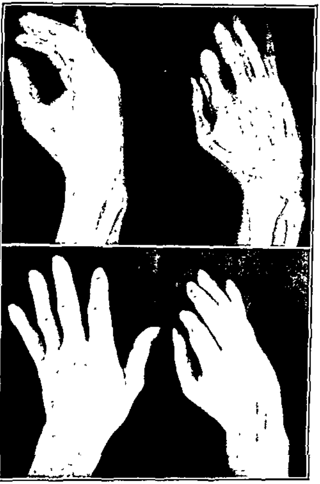


FIG 178 —Hysterical deformity of the right hand of eleven months' duration Roentgenograms of the involved hand show marked diffuse secondary bone atrophy from disuse. (Boland, courtesy of Ann Rheum Dis)

patients with psychogenic rheumatism are introverts; some are dynamic extroverts and those displaying gross hysterical reactions are often frank exhibitionists

Precipitating Emotional Factors.—Multiple interviews may be needed to ferret out those environmental factors or external events which may have precipitated a deep-seated emotional conflict. It is only by following the sequence of happenings pri
found in some instances
often cause the patient to

PART III

FACTORS INFLUENCING THE ONSET OR COURSE OF THE RHEUMATIC DISEASES

CHAPTER 25

PSYCHOGENIC FACTORS IN RHEUMATIC DISEASE

By Edward W. Boland, M. D.

Psychologic factors in rheumatic conditions have attracted considerable attention in recent years Inquiry has proceeded in two main directions. (1) Attempts to determine whether personality anomalies and emotional states play significant parts in the etiology, predisposition, precipitation and clinical course of undoubted organic disorders such as chronic arthritis, (2) investigations of a large group of cases with ill-defined musculoskeletal disabilities characterized by subjective complaints only, and whose organic nature is questioned by some.

PSYCHOGENIC FACTORS IN RHEUMATOID ARTHRITIS

There are many theories concerning the cause of rheumatoid arthritis No one of these can be fully accepted because each lacks sufficient proof (See Chapter 8, p. 122.) Failure to distinguish sharply between etiologic

including infections of various types, physical exposure, local joint trauma, debilitating states and emotional disturbances may precipitate or aggravate the disease

Before the advent of bacteriology and the many infectious theories concerning the etiology of rheumatoid arthritis, much was written regarding the neural theory.[7] Adherents to this idea postulated the existence of lesions in the central nervous system (chiefly because of symmetrical joint involvement), and some attributed the onset or exacerbations to emotional disturbances More recently many authors have recorded examples where rheumatoid arthritis has developed immediately following emotional shock and where the clinical course has been influenced by anxiety, worry and other psychologic factors [11,12,13,14,21,22,23,26,28]

Direct temporal relationships between the onset of arthritic symptoms and emotional crises are repeatedly observed by physicians who care for patients with rheumatoid arthritis. Sometimes exacerbations and remissions may run parallel to changes in the environmental stress under which the patient lives. Cobb, Bauer and Whiting[7] studied the psychiatric

or present, sometimes provides a suggestion to the psychoneurotic patient

Table 18: Tabular Differentiation Between Psychogenic Rheumatism and "Fibrositis"

	Psychogenic Rheumatism	"Fibrositis"
Patient's attitude	Tense, apprehensive, subjective, often antagonistic or defensive	Usually objective and co-operative
Chief symptoms	Often vague On analysis "pain" may consist of sensations such as burning, tightness, fullness, numbness, weakness, tingling pressure, or "queer feeling" Muscles and joints may be "tense" rather than stiff	Usually definite Consist of aching, soreness and stiffness
Factors influencing symptoms	Symptoms in general fluctuate with changes in internal or mental environment tend to be worse during or following emotional stress, and temporarily relieved by mental distraction (theater, social gathering, sports event, etc)	Symptoms fluctuate with changes in external or physical environment made worse by physical inactivity and damp weather and ameliorated by dry warm weather, physical activity and local heat
Degree of disability	Variable but may be severe and are disproportionate to physical findings Disability may be greater than with organic joint disease	Usually not marked—symptoms more of a "nuisance"
Constancy of complaints	Tend to be constant, "had all the time" or "getting worse" Exercise almost always aggravates	Subject to exacerbations and remissions, may last days or weeks then disappear Intensity varies during day, worse in morning and after physical inactivity, better after exercise
Location of symptoms	Often not anatomical, may "skip here, there and everywhere"	Anatomical If symptoms migrate they do so with respect for anatomy
Response to examination	Often overreacts, is tense and resistant, a "touch-me-not" attitude	Co-operative Mental distraction is without effect
Response to treatment	Variable, often nothing relieves Salicylates frequently ineffective and local heat may make worse, alcoholic beverages relieve temporarily	Temporary relief from salicylates and physiotherapy
Associated functional complaints	Almost always present, i e, nervousness, tremulousness, cold moist hands, insomnia, globus hystericus, headaches, sighing respirations, bizarre postures and limps, etc	Usually minimal or absent
Positive evidence of psychopathology	Present, i e, psychoneurotic predisposition, precipitating emotional conflict, underlying resentment or frustration, coexisting anxiety, depressive hypochondriacal or hysterical reaction	Usually absent

and social factors in 50 patients with rheumatoid arthritis, a life chart being used to synchronize these factors with the clinical course of the arthritis. They found that a close temporal relationship existed between life stress and exacerbations of the disease in 31 (62 per cent) of the 50 cases. In a "control" group, hospitalized because of varicose ulcers, only 12 per cent showed similar relationships. The conclusion was drawn that environmental stress, especially poverty, grief and family worry, bore more than a chance relationship to the onset and exacerbations of the disease. Among the male patients financial worries or lack of work constituted the most common stresses. Among female patients the major burden was also *financial—because of the husband's inability to find work, often followed by* illness in the family and its attendant strain of nursing, using savings, borrowing money or recourse to relief. Family worries, loss of a parent or spouse, marital unfaithfulness, living with in-laws and marital discord also played important rôles in some

The relation of emotional factors to rheumatoid arthritis was studied in 25 cases by Patterson and associates.[24] About half of these had experienced [illegible]

of this mechanism in the development of arthritis; but they were led to the [illegible]

are often emotionally unstable before the onset of their illness. He has stressed the importance of exposing the patient's personal beliefs, resentments, conflicts, hatreds or fears. He feels that if people would cultivate [illegible]

psychosomatic diseases, although he believes that there is a definite genetic element involved also.[14,15] He contends that patients with this disease fit into a particular personality pattern: they show emotional inhibition, are rather [illegible]

is asso [illegible]

they tend to lead quiet lives (as [illegible]
sessional trends as being meticulously orderly, punctual, tidy and clean; in addition they are over-conscientious, are slaves to routine and possess a [illegible]

which influences the localization of somatic symptoms. In cases of superimposed and residual psychogenic rheumatism an immediate example of disability is suggested. In cases of pure psychogenic rheumatism examples, such as arthritic invalidism in the immediate family or a personal experience of traumatic or inflammatory joint disease in the past, may often be elicited. If examples of stomach trouble existed instead, it is probable that functional dyspepsia rather than psychogenic rheumatism would have developed in response to the same emotional stress. The back and lower extremities served as favorite sites for localization in soldiers. The predominant symptoms were located in these regions in 76 per cent of soldiers with psychogenic rheumatism studied by Boland and Corr.[4] This suggested to them that the somatic fixation in the back and lower extremities bore an expedient relationship to the attempted solution of an emotional conflict [illegible] "as these structures are synonymous

[illegible] fibrositis" is the principal rheumatic condition from which psychogenic rheumatism must be distinguished. Because the diagnosis of this condition is based largely on the qualitative characteristics of subjective complaints, differentiation may require a detailed analysis of symptoms The main differentiating points, as listed by Hench and Boland,[19] are summarized in Table 18

BIBLIOGRAPHY

1 Boland, F W Calif Med, 26, 273, 1948
2 ——: Calif and West Med, 40, 7, 1944.
3 —— Ann Rheum Dis, 6, 195, 1947.
4 Boland, E W, and Corr, W P · Jour Amer Med Assn, 123, 805, 1943
5. Boland, E W, and Headley, N E : Ann West Med. and Surg, 2, 289, 1948
6 Boland, E W, Stephens, C. A L. and Hench, P. S : (To be published)
7 Cobb, S, Bauer, W and Whiting, I. Jour Am. Med Assn, 113, 668, 1939
8 Ellman, P, Savage, O A, Whittkower, E, and Rodger, T F.· Ann Rheum Dis, 3, 56, 1942
9 Flind, J, and Barber, H. S Quart Jr Med, 14, 57, 1945
10 Gregg, D [illegible]
11 Halliday [illegible]
12 —— [illegible]
13 —— [illegible]
14 —— Practitioner, 150, 6, 1944
15 —— Personal communication, 1945
16 —— Lancet, 2, 692, 1943.
17 —— Ann Int Med, 15, 666, 1941
18 [illegible] (1946)
19 [illegible]
20 [illegible] berg, R H, [illegible] Rosenberg, [illegible]
21 [illegible]
22 Jelliffe, S E, and White, W. A : Diseases of the Nervous System, 6th ed, Philadelphia, Lea and Febiger, 1935
23 [illegible] Jour Med, 214, 576, 1936
24 [illegible], and Freyberg, R H · [illegible]
25. [illegible] 47
26 [illegible]
27 [illegible]
28 [illegible]
29 [illegible]

object, resentment concerning superiors, frustration at being jilted, etc

...worrisome kind, 4 of
... mood, in general,
...rther evidences of
... of the 14 females
...d 3 of the 6 males
demonstrated marked fixations to their mothers or sisters

Gregg has made the interesting observation that psychotic individuals

dream states (in their variations), while the arthritic (through somatic or physical pathways) develops through joint disability

Whereas the extreme view—that rheumatoid arthritis represents a psychosomatic illness—offers interesting speculation, it is held by only a few students of the disease. Most authorities agree that emotional upsets may

PSYCHOGENIC FACTORS IN FIBROSITIS

Unfortunately, the term fibrositis has often been loosely used and ill-defined in medical writings Too frequently it has been applied in practice as a "cover-up" label to explain or to dismiss vague musculoskeletal symp-

ness referable to muscle regions and joints show undoubted psychologic

labelled with the diagnosis of rheumatism (fibrositis, lumbago, neuritis and sciatica) and concluded that 39 per cent were actually incapacitated because of psychoneurosis Ellman and associates[8] studied 50 cases diag-

CHAPTER 26

FOCAL INFECTION IN RHEUMATOID ARTHRITIS

By WALLACE GRAHAM, M.D.

Despite a complete absence of controlled observations pointing to its validity, the theory of focal infection still ranks first in the minds of many physicians and dentists when confronted with a rheumatic patient. In many instances the *removal of teeth and tonsils* is not only the first but the *only treatment recommended* The widespread application of this theory has resulted in an appalling loss of oral and other tissues without commensurate benefit to the patient. On the other hand, it would be unwise if the pendulum swung too far in the opposite direction to a point where infection is entirely overlooked and considered to be of no significance Such an attitude is equally unjustified, the eradication of proven infection in the arthritic patient is indicated the same as it would be if the patient did not have arthritis. *As long as the cause of rheumatoid arthritis is unknown, the possible role of as yet unknown infectious agents remains a possibility*

In discussing the role of focal infection in rheumatoid arthritis, two all important facts must be kept in mind.

1. The etiology of rheumatoid arthritis is as yet unknown
2. The basis for the theory of focal infection in rheumatoid arthritis is still entirely theoretical

Definition.—Billings[1] defined a focus of infection as "a circumscribed [illegible]

(*1*) that there exists or has existed a circumscribed lesion or focus, (*2*) that the lesion is of bacterial nature and, as such, is capable of dissemination, (*3*) that dissemination from the focus has resulted in systemic infection or infection of other contiguous or non-contiguous parts

History.—The concept of focal infection is by no means new. Two [illegible]

mouth had relieved patients of rheumatic troubles in the joints. The more enthusiastic supporters of the theory have even linked the pangs of toothache suffered by Bobbie Burns, and which, no doubt, gave rise to his "Address to the Toothache," with the severe rheumatism which is said to have caused his early death. Rush (1818) published observations made as early as 1801, which suggested the curative effects of removing infected teeth in patients with various general diseases. Following this the idea lay practically dormant for almost 100 years until William Hunter again brought it to the fore.[10] In addressing the students at McGill University on October 3, 1910, Hunter launched the theory of "oral sepsis" which

Because both primary fibrositis and functional musculoskeletal disorders usually present only subjective symptoms, without objective findings, differential diagnosis may be difficult at times. Differentiation and a correct assay of the problem may become a tedious task when fibrositis coexists with a pronounced functional overlay. Hench and Boland[19] believe, however, that the two conditions may be separated clinically if a detailed inventory of the qualitative nature of the complaints is made. In general, primary fibrositis places its victims at the mercy of changes in external environment: Thus weather, heat, cold, humidity, rest, exercise, etc., characteristically influence most of them for better or worse. On the other hand a psychogenic musculoskeletal disorder generally puts its victims at the mercy of changes in internal environment: Thus their symptoms may vary with mood or psyche, pleasure, excitement, mental distraction, depression, worry or fatigue. A more detailed differentiation is given below under "Psychogenic (Psychosomatic) Rheumatism."

PSYCHOGENIC (PSYCHOSOMATIC) RHEUMATISM

(Psychoneurotic Musculoskeletal Complaints)

Although physicians are familiar with the fact that functional symptoms or symptom complexes may be expressed in various other systems, they are not so familiar with the fact that disabilities of the locomotor system frequently result from psychic causes. Functional musculoskeletal symptoms are frequently mislabelled as arthritis or some other organic disease. Thus, by failing to recognize the true nature of the complaints and by treating the patient for arthritis, the disability may be fostered rather than alleviated.

A name, entirely satisfactory to all, has not yet been suggested to describe the musculoskeletal disabilities which may result from psychoneurosis. The term psychogenic rheumatism is objected to by some rheumatologists and psychiatrists because it is claimed that the word rheumatism is too vague and already surrounded by enough opprobrium without linking the word psychogenic to it.[29] Others contend that functional muscle and joint complaints are often vague too, and that the word psychogenic at least points to the origin of symptoms.[3] The word psychosomatic is not entirely [illegible]table because more and more it is being used to imply that actual or-

[illegible] being

mani-

r daily

[illegible] For internists and ortho-

found in organic disease. In practice, therefore, recognition of the quantitative characteristics of the bodily complaints comes first and an exploration of the psychologic mechanism comes later. If its limitations are under-

fired the imagination of many who were seeking a cause for a multitude of obscure diseases. *The widespread effect of this new theory may readily*

recognition and exploitation of its great resources. . . . The chief seat of that sepsis is the mouth. . . . No one has probably had more reason than I have to admire the sheer ingenuity and mechanical skill constantly dis-

depression up to actual lesions of the cord, of chronic rheumatic affections, of kidney disease are those which owe their origin to or are gravely complicated by the oral sepsis produced *in private patients by these gold traps* of sepsis."

It is interesting, however, to note that in the 10 case histories presented by Hunter in this historic address, not one was suffering from any rheumatic ailment.

Hunter's enthusiasm was quickly matched by that of Billings (1913) in America.[16] Following a study of seventy patients with rheumatoid arthritis, he stated, "*It has been found that the focal disease is usually located* in the head. Most frequently there was a chronic streptococcal focus of the faucial tonsils; but chronic alveolar abscess and chronic sinusitis due to streptococcus infection were an occasional cause. . . . There can be no question that chronic gonorrheal infection of the seminal vesicles may cause systemic disease, especially arthritis. Streptococcus infection of the seminal vesicles may also cause systemic infection and especially arthritis deformans."

Further confirmation came fr

teeth are relatively

Following these dramatic presentations, it is little wonder that a plausible theory reached the status of an accepted fact and that physicians and dentists around the world, frustrated in their attempts to cure arthritis, ... removable areas of infection.

organism The many

theories proposed in regard to the etiology of this disease only serve to

and other areas could lead speculation. Several possibilities have been considered—

(*1*) The bacteria might be transported via the blood stream to other organs.

(*2*) Bacterial toxins could be carried to the joint and other tissues

(*3*) The protein fraction of the bacteria could act as an antigen and sensitize the joint tissues so that additional bacteria or protein coming

venously with heat-killed streptococci, and bacteria were injected intravenously into these rabbits at a later date, it required a smaller number of organisms to produce arthritis in the previously injected rabbits than in normal animals. In another group of experiments, small amounts of heat-killed streptococci or nucleoprotein fraction were injected into the right knee-joint of a number of rabbits, when these animals were later injected intravenously with live cultures of the same bacteria, the previously injected joints were much more susceptible to infection than other joints injected with heat-killed streptococci or horse serum. But Collins (1949)[4] has pointed out that the rabbit is so easily sensitized and so easily given arthritis that the results of rabbit experiments are not always applicable to other species, and the critical evaluation of rabbit experiments is made more difficult by the extreme ease with which different procedures will provoke arthritis Collins goes on to state that, in spite of the evidence suggesting that rheumatoid arthritis arises from an infection, neither the nature nor the modus operandi of the infective agent has yet been discovered He groups under four headings the various theories which have been advanced to explain the relationship between infection and rheumatoid arthritis

1 *Virus Infection* —Although the theory is attractive, attempts to recover a virus from the affected joints have failed.

2 *Absorption of Toxins from Focal Infections* —The theory that bacteria in remote sites may elaborate diffusible exotoxins which reach the joints by way of the blood stream and cause arthritis is unlikely, as the pathological changes in the joints do not resemble any lesion which is known to be associated with a simple bacterial toxemia

3. *Tissue Hypersensitivity* (*Allergy*) —Tissues which have been sensitized to a particular antigenic foreign protein show an exaggerated inflammatory reaction when they are subsequently exposed to amounts of the same antigen too small to affect normal tissues The resultant histological changes have certain features in common with rheumatic fever and other human diseases Hypersensitivity may play a part in the production of rheumatoid arthritis

4. *Hematogenous Bacterial Infection of the Joints.*—Although Collins[4] failed to obtain positive cultures from the blood and joint exudates in eighty cases of rheumatoid arthritis, he states that the possibility of bacteremic joint infection initiating the disease has not been excluded. In his experiments on swine, the inflammatory tissue was found to persist after the disappearance of the bacteria from the joint fluid and probably also after sterilization of the synovial tissues.

In summary, Collins points out that the available evidence seems to show that rheumatoid arthritis is neither a pure hypersensitivity disease nor an ordinary bacterial arthritis. To explain the arthritis, he advances

their economic status or disrupt their social ties in a vain attempt to seek a climate that cures It may be possible, in the future, by means of humidity and temperature control in the home, to stabilize better the environmental factors at home [4]

BIBLIOGRAPHY

1 BAZETT, H C Personal communication.

1940
5 HILL, D F, and HOLBROOK, W P Clinics, 1, 577, 1942
6 HILL, L: Brit Med Jour, 2, 276, 1939

19, 1939

17 RENTSZHLER, E B, VANZANT, F R, and ROWNTREE, L G Jour Am Med Assn., 92, 1995, 1929
18 Report on the Epidemiology of Rheumatic Infection in South Australia, Med Jour Australia, 1, 461, 1940
19 SEEGAL, D, SEEGAL, E B C, and JOST, E L Am Jour Med Sci, 190, 383, 1935
20 SHANDS, A R Unpublished data, 1944
21 TEGNER, W S Ann. Rheum Dis, 1, 249, 1939

a theory based upon the combination of minimal metastatic bacterial infection with tissue hypersensitivity. "An individual suffers a series of minor infections, *e. g.*, of throat or naso-pharynx, in the course of which a few bacteria invade the blood stream. Alternatively, he acquires a chronic focus of bacterial infection which from time to time discharges a few bacteria into the blood. The organisms are rapidly removed from the blood and at no time are they present in sufficient numbers to cause conventional metastatic infective lesions. The joint tissues, however, become sensitized by these repeated inoculations, the sensitization increasing in degree until eventually one bacteremic episode results in a manifest arthritis. The course of events thereafter may be subject to a great many influences, about which we have little information. It is clear, from the study of the tissues in both animal and human arthritis, that this is no ordinary pattern of inflammation." However, Collins goes on to state that the theory, well founded on analogies, has not been substantiated by direct evidence in man.

Organisms actually do pass from foci of infection into the blood stream. It has been definitely shown that in patients with pus pockets surrounding their teeth, the extraction of such teeth or the mere act of chewing hard candy may be followed by a bacteremia, usually streptococcus viridans, but the blood stream infection is transient and apparently innocuous in those not suffering from congenital or rheumatic heart disease.

The fact that the removal of infected teeth and tonsils does not cure rheumatoid arthritis, does not rul[illegible]
factors in initiating the disease. [illegible]
to Cortisone and ACTH has draw[illegible]
and stimulated a search for a com[illegible]
Brown and his associates have presented evidence that an unusual form of hypersensitivity reaction may be the common factor.[2] They propose that the effect of Cortisone on rheumatic disease may be due to its influence on the immune mechanism. They note that many cases of drug hypersensitivity are clinically indistinguishable from acute rheumatic disease, and suggest that the chronicity of rheumatic diseases, if explained on a hypersensitivity basis, must depend on the persistence of an antigen at the site of continuous antigen-antibody reaction, presumably at the cell surface. As the usual types of bacteria have been excluded, they assume the presence of a virus or virus-like agent. The pleuro-pneumonia-like organisms ("L" organisms) are cited as a possible source of antigen although their role in rheumatic disease cannot be proven or disproven by present laboratory means. However, the specific effect of gold salts on "L" organisms would appear to lend some support.

The effect of various antibiotics on "L" organisms was also studied and it was observed that Terramycin, which produces a more intense exacerbation of rheumatic activity in patients than Aureomycin or Chloramphenicol, was also more effective against "L" organisms in vitro. The therapeutic use of various antibiotics would cause a release of antigen and might induce a dangerously severe reaction unless the antigen was completely elimir[illegible]
Cortisone or [illegible]
combined wit[illegible]
and possibly [illegible]
15, p. 247.)

CHAPTER 28

THE RÔLE OF THE ENDOCRINE GLANDS IN THE RHEUMATIC DISEASES

By John Lansbury, m.d.

That a relationship between endocrine function and certain rheumatic diseases exists is a well established clinical fact which cannot be denied.

and pains of myxedema and of the menopause may also be cited since they respond respectively to the administration of desiccated thyroid substance and estrogens. Another example of the endocrine-rheumatism relationship is the periodic exacerbation of arthritis timed with the menstrual cycle (arthritis intermittens). Other less striking examples of such relationship could be cited also.

The dramatic "anti-rheumatic" action of ACTH, cortisone and hydrocortisone raises the question as to whether subtle disturbances in pituitary and adrenal function may play an etiologic role in the pathogenesis of the rheumatic diseases. However, it seems clear that commercially available *adrenal cortical steroids*, as now used, *do not alter the fundamental disease process*, but act through a *nonspecific anti-inflammatory mechanism* which is *by no means confined to the rheumatic diseases*, and which therefore *sheds no direct light on their pathogenesis*.

In this chapter the author attempts to present the practical application of endocrinology to arthritis, and to indicate current research, theory, and those attempts at hormonal therapy which seem significant. The action of the sex hormones is discussed in the chapter on Pregnancy and Arthritis and the pharmacology of corticotropins and adrenal corticosteroids is dealt with in the chapter devoted to the clinical use of cortisone and ACTH. (Chapter 17.)

THE PITUITARY GLAND AND ARTHRITIS

There is, in general, no obvious relationship between frank pituitary disease and rheumatoid arthritis.

The arthritis of acromegaly was described by Marie in 1886[14] Kyphosis

We have observed numerous instances of Osgood-Schlatter's Disease (separation of the tibial tubercle) in fat boys of the "hypopituitary" type.

Clinical Evidence.—The sharp change in attitude toward the importance of foci of infection in relation to the rheumatic diseases is reflected in the opinions of many who have observed the application of the theory in many rheumatism clinics A review of American and English papers published from 1940 to 1945 led to the general conclusion that there is no proof that rheumatoid arthritis is an infectious disease.[8]

Another convincing argument that rheumatoid arthritis is probably not an infectious disease was recently reported by Levinsky and Lansbury.[11] They attempted to transmit the disease by injection of synovial fluid from rheumatoid arthritic patients into non-arthritic human subjects, and in

of fatigue, weight loss, low-grade fever, and lymphadenitis, (*2*) elevated blood sedimentation rate and leukocyte counts, (*3*) frequent elevations in streptococcic agglutination and antistreptolysin titers, (*4*) frequent positive skin tests for streptococcic protein fractions But, as he pointed out, these

(*a*) Streptococcic infections of throat, tonsils or nasal sinuses may precede the initial or recurrent attacks

(*b*) Dramatic improvement sometimes follows removal of a septic focus

(*c*) The pathological and anatomical features of lymphoid tissue in tonsillar infection, sinus infection and root abscess suggest that toxic products can be absorbed into the circulation

(*d*) A temporary bacteremia may occur immediately after tonsillectomy or tooth extraction, and after vigorous massage of the gums

Points against the importance of septic foci included

(*a*) Often no infected focus can be found

(*b*) Usually when a focus has been extirpated no dramatic results are produced.

(*c*) Many persons in good health or suffering from diseases other than rheumatoid arthritis may have septic foci in the same situations and of the same magnitude as patients suffering from rheumatoid arthritis

found in 44 per cent of cases of rheumatoid arthritis and in 43 per cent of controls Only 8 patients in the rheumatoid group gave a history of an infection of the upper respiratory tract within three months of the onset of the arthritis The results obtained failed to suggest that infection of the ear, nose or throat play an important part in the etiology of this disease.

Among 239 psychiatric patients Brown[15] found infected foci in the nose and throat in 44 per cent, in the teeth in 37 per cent, and in the urinary tract in 17 per cent, yet none of these individuals had arthritis

However, so little is known of the endocrine disturbance in these cases, that no proof of a definite pituitary-epiphyseal relationship is claimed.

Among the more recent experimental observations dealing with the
mention the work of Edstrom and
nterior pituitary glands 30 minutes
buttocks of rheumatoid arthritics
Clinical and metabolic effects similar to those produced by ACTH followed, but with this important difference: twenty-two patients obtained a remission for 9 months, and 25 per cent for 3 to 4 months. This prolonged effect is difficult to explain on the ACTH content alone (estimated as 1 to 3 mg) especially since heterologous pituitary implants undergo necrosis and absorption in a few days. The effect was enhanced if the pedicle towards the tuber cinereum was included in the implant. Selye[19] in discussing the adaptation syndrome, points out that the somatotropic (growth)

the growth hormone stimulates connective tissues thus acting as an antagonist to the corticotropic hormone (ACTH). So far there is no objective proof of excess growth hormone during stress or of its relation to rheumatoid arthritis Negro[18] reports from Italy on a large series of arthritics (presumably rheumatoids) treated with orally administered extracts of posterior lobe pituitary He claims an anti-rheumatic effect after 1 or 2 months' treatment Psoriatic arthritis was also benefited The Haydus[11] report a prompt reduction of pain and stiffness in rheumatoid arthritis following the injection of 4 units of pitressin The effect disappears in about 24 hours They believe the mechanism of action to be an inhibition of abnormally increased adenosintriphosphatase They point out that gold salts inhibit this enzyme and that salicylates inhibit closely related enzymes The action is not mediated by the adrenal cortex. This

hormone on the connective tissue system is not associated with gamma globulin changes, a fact which distinguishes myxedema from the collagen diseases.

to

pr

have some relation to disturbances in the connective tissue system, and which merit the interest and study of rheumatologists

THE ADRENAL GLANDS AND ARTHRITIS

As in the case of the other endocrine glands, there is no obvious relation between frank clinical disease of the adrenal glands and the rheumatic

The findings of Ellman were similar.[7] "Of the last 100 patients attending the rheumatism unit at St. Stephen's Hospital, London County Council, the duration of whose disease varied from 6 months to 25 years and averaged 6 1 years, many were found to be divested of teeth and tonsils, some with sinuses drained, gall-bladder, appendix, and other organs removed. Yet, in spite of such radical measures in dealing with supposed focal infection, the patient was frequently no better but, on the contrary, despondent and lowered in morale." Ellman calculated that no more than 3 per cent of these 100 cases had genuinely benefited from such treatment. He also pointed out that, "Similar enthusiasm has even been extended to the

greed that focal
heumatism has
is surely need



Fig 179 —*A*, Deep pocket under overhanging filling; *B*, pyorrheal pockets around upper incisor, *C*, area of active osteomyelitis around impacted third molar (Haden)

Short and Bauer[13] agreed that the expensive combing of a patient for sources of infection is seldom warranted. They stressed the importance of having the physician carefully interpret and correlate the findings before acting on isolated areas Frankel[7a] found no correlation between systemic disturbances and the extent of dental restorations in a series of 17,000 persons. Appleton[1]
in those with the g
x-ray examination
as a cause of systemic disease is far from convincing.

Some edentulous patients harbor retained roots of which they are not aware, but not all of them are infected. Many are just as innocuous as a piece of shrapnel or wire suture and do not necessarily constitute an area of so-called residual infection. Sorrin[14] commented on the frequency of
rthritic patients, but reached the
ver, constitute a focus of infection
l for this lesion alone. Similarly,
unded by an area of infection, are
as far as rheumatoid arthritis is
erefore, be given the same critical

consideration.

arthritis and rheumatoid arthritis is rare among hypertensives.[23] In so far as hypertension may be considered as being due to adrenal overfunction, this suggests, but does not prove, that adrenal function is low in rheumatoid arthritis. Occasionally, in very depleted rheumatoid arthritics one may find skin pigmentation, in some respects resembling that of Addison's disease, which can be reversed by cortisone. However, if adrenocortical function is lowered in rheumatoid arthritis, it could just as logically be the result as the cause of the disease.

In Addison's disease, where there is a diminution of all corticosteroids, the administration of desoxycorticosterone acetate may produce joint pain and even effusions. But, an excess of desoxycorticosterone has not been detected in rheumatoid arthritis, neither has this hormone been positively identified in the adrenal cortex. No final statement can therefore be made

than the disease itself. Thus, in lobar pneumonia, the fever, leukocytosis, cough and lobar consolidation constitute the body's defense against the pneumococcus. It may well be that the inflammatory features of rheumatoid arthritis should also be regarded as the body's defense rather than as the disease itself. That this defense is, in part at least, regulated through *the pituitary-adrenal axis cannot be denied. Neither is it unreasonable to* suppose that other endocrine regulators of the body defense (adaptation) may exist.

At present it seems certain that ACTH, cortisone, and hydrocortisone act by suppressing the body's reaction to disease rather than by suppressing the disease itself. Their effect therefore does not explain the cause of rheumatoid arthritis On the other hand, the question as to whether or not the rheumatic diseases are primarily due to a disturbance in the adaptation or defense mechanism itself is, of course, a matter of great importance, but it cannot be settled on theoretical grounds alone; it must await the detection of actual hormonal disturbance in cases of rheumatoid arthritis This is, unfortunately an exceedingly difficult task. The fact that stress of various kinds, especially emotion, is a precipitating factor in the majority of cases of rheumatoid arthritis suggests that the resulting disturbance in the pituitary-adrenal axis *is* a factor in the disease.

After considerable deliberation, the author has decided against an attempt to review *the theoretical aspects of the Alarm Reaction and the* General Adaptation Syndrome[19] in relation to rheumatism. Any such review would probably be out of date by the time this work is published, furthermore, any conclusions reached would be highly tentative, theoretical ... the ... m in ... der- ... nore

theory in this field.

... 30 steroid hormones ... f the great difficulties ... ether these hormones ... ther the majority of

them are the products of chemical manipulations attending their extraction,

In a very few patients an arthritis may begin with a rather sudden onset usually confined to one joint. In such individuals the removal of a focus of infection may be followed by disappearance of the arthritis. These

nephritis, iritis, and endocarditis have been observed shortly after the onset of an acute tonsillitis or sinusitis or alveolar abscess. In such patients it is sound clinical practice to treat or remove the infected areas as soon as possible.

In the Primer on the Rheumatic Diseases prepared by the American Rheumatism Association (1949), the situation is summarized as follows "No one now believes that the removal of an infected focus will alter the course of rheumatoid arthritis, and extraction of teeth, tonsils, gall-bladders and pelvic organs is to be condemned unless they are so diseased that they should be removed for their own sakes Indeed, wholesale removal of teeth usually is not only useless but actually harmful to the patient through interference with nutrition "

Rheumatoid Arthritis Precipitated or Aggravated by Gonorrhea —Hench and Boland[9] have pointed out that rheumatoid arthritis can be precipitated by a gonorrheal infection just as it can be precipitated by tonsillitis or some other acute infection Also a mild intermittent or quiescent rheuma-

genital (non-articular) gonorrhea. Many of these cases have been er-

penicillin

eumatoid

arthritis rarely results in any beneficial effect It may *lessen the load of infection and thus improve the patient's general health* but naturally will have no effect upon pre-existing contractures, adhesions and ankyloses

However, in the rare situation where arthritis begins shortly after a tonsillitis, sinusitis or periapical infection, it is good clinical practice to treat or remove the infected area as soon as possible

What attitude should then be taken concerning definite foci of infection in the patient with rheumatoid arthritis? It would seem a reasonable

etc are no more in

le *Proper attention*

'ent with rheumatoid

arthritis the same as they would be attended to if the patient did not have arthritis. The patient should be told that the *removal of such infection* will probably *not influence the course of the arthritis* but the procedure is recommended as a *general health measure.* Should the patient be suffering from rheumatic or congenital heart disease, it is essential that pre- and post-

or are metabolites and cleavage products resulting from body metabolism

The adrenal cortex is composed of three histologically distinct layers

as its acetate by the name of DOCA, is believed to be the same or similar to the naturally occurring mineralocorticoids, although it has not been positively identified as such in the zona glomerulosa. It is thought not to be under the control of ACTH in humans It increases the flow of fluids across semi-permeable joint membranes—an action which is inhibited by cortisone.

The middle layer, or zona fasciculata is probably the site of formation of the glucocorticoids, mainly in the form of 17-hydroxycorticosterone, known also as Compound F or hydrocortisone.

Much less is known regarding the androgenic steroids which are believed to arise from the innermost layer or zona reticulosum and which are chemically distinct from testicular androgens Only when adrenal androgens become available for clinical trial will their role in disease be clarified

The output of glucocorticoids is stimulated by ACTH, and the pituitary gland in turn may be stimulated to pour out ACTH either by direct

of ketosteroids, formaldehydogenic steroids and glucocorticoids by group reactions have produced interesting but sometimes conflicting results An increased excretion of 17-ketosteroids has been reported in cases of rheumatoid spondylitis, but is not constantly present In gout the 17-ketosteroids are decreased in the urine, possibly on account of an abnormal androgen which is not converted to 17-ketosteroids as it should be Low 17-ketosteroid outputs have also been reported in scleroderma, and testosterone has been advocated in the management of this disease

Staub *et al* [21] found a slight rise in 11-oxycorticosteroids in 14 cases of rheumatoid arthritis, but Venning *et al* [24] found the urinary secretion of glucocorticoids and of total corticoids to be depressed in chronic rheumatoid arthritis although elevated in acute, severe cases

Probably the work of the late Dr Dobriner[7] is the most significant in this field He found the total secretion of steroid metabolites to be consistently at the lower limits of normal in a series of 5 cases of rheumatoid arthritis. He studied the excretion pattern of a large number of steroid metabolites in normal people. While there were individual variations among the various metabolites, these occurred within certain limits so that he was able to establish a normal steroid excretion pattern or profile, in 30 healthy individuals.

In rheumatoid arthritics there was not only a quantitative reduction in total steroid output, but in 4 of 5 cases, an abnormal steroid, *17-hydroxy pregnanolone* was detected. This steroid has also been detected in cases of adrenal virilism and adrenal tumors. The excretion of this abnormal

operative penicillin therapy be instituted to counteract the bacteremia and possible bacterial endocarditis which may follow the operative procedure

Summary.—It is now generally agreed that the removal of the common foci of infection has little if any effect upon the course of rheumatoid arthritis. Over the years this very plausible theory ultimately reached the status of an accepted fact and thousands of rheumatic patients were doomed to an edentulous existence due to the lack of critical and scientific approach to a subject whose basis was entirely theoretical.

Acceptance of the theory must still await controlled evidence that foci of infection play a part in the etiology of rheumatoid arthritis. After reviewing the unfortunate results of the application of this theory over a period of 40 years, it is astonishing that it still leads to the wholesale removal of teeth and tonsils as a "cure" for many forms of rheumatism

BIBLIOGRAPHY

1 Appleton, J L T, Jr · Bacterial Infection, Ed 3. Phila., Lea & Febiger, 1944
1a Billings, F. Jour. Am Med. Assn, 61, 819, 1913
1b Brown, C H Am Jour. Med Sci., 199, 539, 1910.
2 Brown, T. McP, Merchant, W R, Wichelhausen, R H., and Robinson, L B · Am Jour Med Sci, 221, 618, 1951
3 Cecil, R L: Proc Mayo Clinic, 15, 556, 1940
4 Collins, D H. The Pathology of Articular and Spinal Diseases, London, Edward Arnold & Co, 1949
5 Davidson, L S P Trans. Royal Med-Chir. Soc, Glasgow, 2, 6, 1943.
6. Davidson, L S P, Duthie, J J R, and Sugar, M · Ann Rheum Dis, 8, 205, 1949
7. Ellman, P: Proc Royal Soc Med, 40, 332, 1946
7a Frankel, L K, *Dent Cosmos*, *66*, *35*, *1924*.
8 Hench, P S *et al* Ann Int Med, 28, 66, 309, 1948
9 Hench, P. S, and Boland, E W · Ann Int Med, 24, 808, 1946
10. Hunter, W Lancet, 1, 79, 1911.
11. Levinsky, W J, and Lansbury, J. Proc Soc. Exper Biol. and Med, 78, 325, 1951
12 Rosenow, E C *Dent Cosmos*, *59*, *485*, *1917*.
13. Short, C L, and Bauer, W New England Jour. Med, 227, 442, 1942
14 Sorrin, S Jour Dent Res, 20, 513, 1941

THE THYROID GLAND AND ARTHRITIS

ship to rheumatism. The majority of patients with chronic arthritis have normal thyroid activity and the occurrence of hypothyroidism in such patients is a coincidence.

Hypothyroidism may present itself by such apparently unrelated symptoms as hoarseness, depression or menorrhagia, but a certain number of hypothyroid patients seek medical aid on account of "rheumatism" and the astute rheumatologist therefore will be constantly on the look-out for masked myxedema masquerading as "arthritis." The type of pain is not of itself diagnostic. It may be associated with a subjective sensation of stiffness and coldness. It is diffuse and vague in location and is more apt to be confused with fibrositis than with arthritis Since the symptoms are readily abolished by the oral administration of desiccated thyroid substance,

Hypertrophic Pulmonary Osteoarthropathy and Lowered Thyroid Function.—Rynearson and Sacasa reported a case of hypertrophic pulmonary osteoarthropathy which developed coincidentally with the onset of postoperative *myxedema*[18] *Cushing*[5] *and Thomas*[22] *reported similar cases* occurring eight years and eight months respectively after subtotal thyroidectomy.

Cortisone Withdrawal Syndrome and Thyroid Function.—In this dis- ... (see Chapter 17) there is good

... y ... e ... r

elevated serum cholesterol. The author has observed some subjective ... cases of the cortisone

... co-exist, and, there is ... seases Occasionally a fulminating form of rheumatoid arthritis may bear some superficial resemblance to hyperthyroidism on account of fever, weight loss and muscular weakness.

RHEUMATISM AND THE MENOPAUSE

Disturbed Physiology.—At the time of the menopause the ovarian steroid hormones are gradually withdrawn, and, *in the case of an artificial* menopause, abruptly so. One of their many functions is periodically to inhibit the anterior lobe of the pituitary gland, so that, at the menopause, the pituitary for a variable length of time *secretes an excess of gonadotropic*

CHAPTER 27

CLIMATE AND THE RHEUMATIC DISEASES

Revised by W. PAUL HOLBROOK, M.D.

THAT climate has an important influence on the incidence and recur-

Cold, dampness and chilling *have been condemned for centuries by patients nd physicians as predisposing to the onset or aggravating rheumatic diseases.*[16] owever, there are very few precise data to support such a contention limate, weather and atmospheric conditions are made up of many varible components such as barometric pressure, humidity, temperature, ind velocity, atmospheric electricity, sunshine, variation in rainfall, oudiness, storminess, etc

It has been noted that *barometric pressure* influenced the symptoms of ost arthritic patients more consistently than humidity or temperature, it it did not influence all arthritic patients in the same manner[17] Some these felt better when the barometer fell while others were better when rose. *Humidity* seems to play a definite rôle in determining the sympoms of many arthritic patients *Most rheumatic individuals feel better on ot dry days* or on cold dry days and worse on hot humid days or cold amp days

Rheumatic pain is often increased in connection with atmospheric *changes, is is noted especially with a fall of barometric pressure as occurs before thunderstorm*[12] Rheumatic patients have thus become known as weather ophets. It has been suggested that such pains may be caused directly y the increased sensitiveness of the sensory nerves, arising from variation the electrical ion concentration of the air in a negative direction[3]

It has also been shown that there may be enormous shifts in blood

orse in what we consider to be bad weather In one series in which the ffects of the weather were studied on 367 patients, no consistent relationhip was found between the symptoms and temperature, atmospheric lectricity or humidity when each factor was considered alone.[17] Seventywo per cent of the time, the patients felt worse as the barometric pressure

and possibly other hormones. Eventually equilibrium is re-established, possibly by compensatory activity of the adrenal cortex, and the menopausal symptoms disappear. The Silbergs[20] showed that pituitary implants (and administration of other hormones) caused joint cartilage to proliferate in young animals whereas in old animals the attempted cartilage response ended in degeneration, erosion and pannus formation This suggests (but does not prove) that excess pituitary hormones may be a factor in causing arthralgia and osteoarthritis

However, before too many conclusions are drawn as to the importance of sex hormones in menopausal rheumatism, one must consider the psychosomatic aspects of the climacteric. The menopause is not only a time of emotional instability which may of itself lead to exaggeration of minor aches and pains, but it often coincides with devastating changes in a woman's environment. It is at this period that children may become self-sufficient or leave home and that husbands may become less faithful The woman whose interests have been limited to her home and family may find herself rather suddenly no longer needed and unable to develop new goals or satisfying interests Such emotional storms, aside from producing psychoneurotic symptoms, may be one of the trigger mechanisms which set off the train of events ending in rheumatoid arthritis (*See also* chapter on Psychogenic Factors in Rheumatic Disease, p 497)

Menopausal Arthralgia.—Diffuse aches and pains in muscles and joints are common at the time of the menopause, and, when there are no objective signs of true arthritis, such as joint swelling, heat, redness, tenderness or

estrogens such as stilbestrol and are not to be confused with those due to true arthritis occurring at the menopause

There is no proof that these pains are directly due to the hormonal changes of the menopause, although we might consider this to be most likely. Osgood has favored this view.[17] Others would explain the symptoms on the basis of generally increased awareness of bodily sensations during the menopause. Certainly we have all seen diffuse aches of an apparently psychogenic nature, at any age, which are relieved by psychotherapy. (*See* chapter on Psychogenic Factors in Rheumatic Diseases)

Until the etiology has been settled, we may be content, from a practical point of view, to distinguish menopausal arthralgia from arthritis and to know that often it can be symptomatically benefited by estrogens.

Arthritis at the Menopause.—Arthritis not uncommonly begins at the time of the menopause. Fletcher notes the onset of 9 7 per cent rheumatoid and 18 per cent of osteoarthritis at the time of the menopause in a series of approximately 250 cases of each.[9] When such arthritis is frankly rheumatoid or frankly osteoarthritic, no unusual problem in diagnosis or management is raised and, when both rheumatoid and osteoarthritis clearly coexist, the term "mixed arthritis" is justified, if qualified by including the relative severity of each form.

Post-Menopausal Osteoporosis.—Averaging ten years after the physiologic menopause, women are prone to develop osteoporosis Since osteoporosis occurs relatively seldom in men of the same age group, and since these patients can be made to retain calcium by intramuscular injections of

fell and improved as it rose; 21 per cent of the time, the reverse occurred Seven per cent of the time, no correlation could be noted. Storms seem to be *associated with increased pains more than 90 per cent of the time*, but patients vary from time to time in the response to these factors.

Segal studied his patients' subjective response to changes in barometric pressure in New York and found no confirmation of the usual impression that the rheumatic patient acts as his own barometer.[19]

Tegner found that the incidence of rheumatic disease fell off definitely and sharply as one approached the warmer climates. Northern European spas had large numbers of patients with rheumatoid arthritis, while those in southern Europe had very few of these individuals.

No one has yet definitely proved that the prognosis of rheumatoid arthritis is materially altered by a warm, dry, climate, although the patient may feel relieved temporarily by a stay there. *Climatotherapy is generally not effectual by itself*, but is combined with rest, psychotherapy, physical therapy and other factors Holbrook and Hill, who have had an extensive experience with arthritic patients in Arizona, concluded that *climate is not a panacea for arthritic patients;* in their experience, unless one employs an intensive program of therapy in addition to rest and sunshine, *chronic arthritis may continue as "a sad and crippling disease even on the Arizona desert"*[5] Unfortunately, many arthritics believe or hope that they will be cured merely by a stay of a few weeks or months in a warm, dry climate, and not infrequently make unjustified financial sacrifices to move temporarily to such a region Rheumatoid arthritis is world-wide in its distribution even though it may be uncommon in some parts of the universe

The effect of *ultra-violet irradiation* or *sunshine* alone is not of itself especially beneficial in the treatment of rheumatoid arthritis; during the acute stage while the patient is febrile or in markedly debilitated patients *with fever, heliotherapy may cause dizziness, further elevation of temperature* and nausea and vomiting.

In the southwestern portions of this country, the barometric pressure is unusually stable and there is a low degree and low frequency of storminess as well as a small amount of rainfall, low humidity, fairly high mean temperature and a high percentage of yearly sunshine. Because of these factors, it has been suggested that this climate may be beneficial to some sufferers from rheumatic disease

In Europe, countries with a high proportion of coastal area feel that "sea air" *is good for the rheumatic patient; those countries with* little coastal area have a disposition to recommend that rheumatic patients get away from the sea; in countries with extensive highlands, there is a tendency to attribute a great deal of importance to altitude as a favorable climatic factor

One finds as much rheumatism on the granite hills of Sweden as on the flat islands of Holland and Denmark. Scandinavian countries usually [illegible] In

patient.[21]

Rheumatic fever is most common in the United States in the early spring and in England, in the late fall. It is common in the cold and wet months of the year.

estrogenic substance, Albright[1,2] believed that the withdrawal of estrogenic hormones was the most important etiologic factor in this post-menopausal syndrome. Other factors are disuse, senescence and possibly faulty diet. *The type of osteoporosis under consideration is not primarily* a disease of calcium and phosphorus metabolism, but rather a failure of osteoblastic activity to keep pace with the normal destruction of bone matrix. The bone matrix which remains is normally calcified. This type of osteoporosis is to be distinguished from that due to hyperparathyroidism in which there is increased bone destruction, and from that due to osteomalacia and Milkman's syndrome where there is failure of calcification of the normal bone matrix.



FIG 180.—Roentgenogram showing advanced post-menopausal osteoporosis Note the biconcavity of the vertebral bodies due to pressure of the intervertebral disks (so-called 'fish vertebrae') and compression fracture of the body of T 11.

In post-menopausal osteoporosis the spine and pelvic bones are chiefly ... This is ... which

the skin.
spine occu
fracture.
lower thoracic and lumbar spine with perhaps some stiffness and

In Hill's series, *90 per cent of patients* (with either rheumatoid arthritis or degenerative joint disease) claimed that they *were weather sensitive*—usually noting increased joint pain, stiffness and swelling when there was a marked change in barometric pressure, storminess, a damp coldness or

e

who have

to impro

and exac

question

at a spec

sibly in Minnesota or Wisconsin) where patients may be equally far from their homes and the worries and cares of their families, and may be equally hopeful on arriving that they will be improved.

Patients with *degenerative joint disease* and with *secondary fibrositis frequently complain of the aggravating effects of dampness and cold,* but much less consistently than individuals with rheumatoid arthritis or primary fibrositis.

Patients with degenerative joint disease and gout are apparently affected

Patients with various forms of arthritis do not act alike in their response to changes in the weather Some patients with rheumatoid arthritis are apparently more disturbed by changes in barometric pressure than by

Arizona, rheumatic patients at times feel better when they spend the winters there, possibly because of the relative constancy in barometric pressure and humidity.[14]

After a careful study of the subject in numerous countries, Tegner concluded that local climate is a relatively unimportant factor in the incidence

play

isease

than those living in temperate zones who may live indoors in poorly ventilated, artificially heated homes *None of Scott's or Shackleton's Arctic explorers developed rheumatic disease despite the severe cold.*[15]

Hill thought that the "*microclimate*" *of the home and working place* was of greater importance than the general climate in initiating rheumatic disease.[6] In his opinion, dampness, cold, atmospheric electricity and cyclonic disturbances were unimportant, but that one should stress the use of warm, well-ventilated houses, warm porous clothing, and the consistent use of daily home methods of physical therapy, supplemented, when possible, by professional physical therapy Edstrom's recent excellent work[16] shows a remarkable effect of a hot environment on arthritis.

varying degrees of rarefaction of the vertebral bodies especially Often there are fractured or crushed vertebrae, or biconcavity of the vertebral bodies due to expansion of intervertebral disks, (fish vertebrae) and herniation of the nucleus polposus through end plates of these bodies (Fig. 180) Although there may be increased calcium excretion early in this disease, the blood calcium and phosphorus levels are normal

In the differential diagnosis, hyperparathyroidism, hyperthyroidism, Cushing's syndrome, Milkman's syndrome, metastatic neoplasms, such as multiple myeloma, and the osteoporosis of sprue and malnutrition are to be considered. Senile osteoporosis may be considered as part of the general atrophy of old age perhaps due to physiologic adrenal cortical failure.

Although Albright induced a positive calcium balance in post-menopausal

recorded) No symptomatic improvement was mentioned

at the menopause, but their mode of action, whether mainly psychogenic or whether by palliation of the hormonal disturbances occurring at this time of life is unknown

The changes of osteoarthritis are degenerative and irreversible and estrogenic therapy therefore cannot 'cure' this type of arthritis At most it might, theoretically, slow up the process by its proved action of suppressing pre-pituitary overactivity. It may be tried, therefore, in the rapidly

effective in fibrositis associated with hypomenorrhoea,[10] or in rheumatoid arthritis in general.

Diethyl-stilbestrol is a cheap, synthetic estrogenic substance which is

Estradiol benzoate, or other ... ecent claims of successful therapy ... atoid spondylitis are discussed in ... and Arthritis. (See p 537.)

THE PARATHYROID GLANDS

There is no known relation between abnormal parathyroid function and arthritis, calcinosis and scleroderma. Rheumatoid arthritis has been

Mills has emphasized that atmospheric turbulence, or storminess, with its accompanying sudden changes in temperature, humidity and pressure constitute a major disturbing factor in certain areas where cyclonic storms prevail. In his opinion, storm changes constitute a major health factor in areas where they are abrupt and frequent, but that the present evidence is too scanty to clearly understand the physiology of their effects.[10] Individuals differ very greatly in their sensitiveness to storm changes.

A high intake of vitamin B has been thought to be of great *importance in protection against severe heat*, 10 mg. of thiamin chloride
shown to be more effective th
the effects of severe heat.[10] I
for thiamine has been found to be twice as high at 91° F. as at 65° F.

It must be emphasized that in the great majority of instances, a mere residence in the southwest will not result in any marked improvement in the patient with rheumatoid arthritis;[7] in other words, *the rheumatoid arthritic should not give up his job, sell his belongings and move to the southwest in the hope that he will be cured by this procedure. Change in climate alone will not result in marked improvement in rheumatoid arthritis.* If the patient moves to a warm, dry area, he should be under the care of a capable physician who has been trained in the treatment of rheumatic diseases.[4]

Although most arthritic patients feel worse in bad weather, and better in good weather, no one has yet determined the factor which may be responsible for such changing symptoms. It is very difficult to evaluate the effect of climate in preventing or ameliorating arthritis because of the multiplicity of factors involved.[4]

Hench,[4] who has had extensive experience in the treatment of rheumatic conditions at the Mayo Clinic, concluded that *most patients must battle their arthritic problems at home and should not be encouraged to endanger*

Summary of Climate and Rheumatic Diseases:

1. All patients with chronic rheumatic disease require first of all expert medical care. *Climate is no substitute for experienced medical supervision.*
2. Rheumatoid Arthritis. A *small percentage* of these patients experience marked relief in a warm dry climate. *The majority, however, are not dramatically affected by climate.* They are, in general, somewhat more comfortable, easier to care for, but just as elsewhere, without adequate medical care, the disease may go on to crippling deformities.
3. Degenerative joint disease, fibrositis, and other forms of chronic rheumatic disease are often *symptomatically improved* by climate change. The effect that any given change of climate will have in such patients is, however, entirely unpredictable and trial-and-error is the only known method at present to determine whether climate will be helpful or not.
4. If a trial period of climate change is considered, not less than *three to six months* should be devoted to change, shorter periods are usually worthless.
5. Climate has a marked influence on the occurrence and recurrence of Rheumatic Fever. In cases where recurrences are frequent or severe, removal of the patient to a warm dry climate may be advisable. (See under chapter on Rheumatic Fever, p. 616).

observed in case of both hyper- and hypo-parathyroidism. Berard found the parathyroid glands to be macroscopically and histologically normal in 20 of 21 cases of "chronic rheumatism."[3] Rarely the bone lesions of hyperparathyroidism if situated near joints may cause arthralgia and limitation of motion and so may be confused with arthritis. The osteoporosis of rheumatoid arthritis and of senile osteoporosis is not due to parathyroid disease. Reduction of parathyroid function by parathyroidectomy and by lowered phosphorus intake has been advocated, but a careful appraisal of these claims fails to establish their value.

THE PROSTATE GLAND

Rheumatoid spondylitis has been thought by some to be due to a disturbed production of phosphatase produced by the prostate gland and conveyed to the spine by venous plexuses connecting the prostatic veins with the prevertebral veins. Desmarais has recently reviewed the inconclusive literature relating to this subject and presented his own studies of the alkaline and acid phosphatase blood levels in a series of 24 cases of ankylosing spondylitis[6] No significant deviation from normal was found This is not surprising when one considers that the disease also occurs in typical form in females.

SUMMARY

As to the *rôle* of glandular products in the treatment of rheumatic diseases, we may safely state that ACTH, cortisone and hydrocortisone have an assured place in therapy. Their value however depends largely upon the skill with which they are used, since they are potentially dangerous drugs when used on a long range basis. In rheumatoid arthritis they must always be thought of as adjuncts to an over-all plan of management in [illegible] Their action [illegible] ter the disease [illegible] doses. *Other* steroids, such as progesterone, also various "pregnancy products" have not so far established themselves as useful anti-rheumatic agents. (*See* next chapter.)

Thyroid substance and estrogens have a small but valuable place in the treatment of diffuse rheumatism.

The great question as to whether rheumatoid arthritis is primarily an endocrine disease remains unanswered. If it be so, then the disturbance must be in some hormonal mechanism as yet undiscovered. Our present knowledge of endocrinology does not explain the nature of the rheumatic diseases.

BIBLIOGRAPHY

1. Albright, F, Bloomberg, E, and Smith, P. H.: Trans. Assn. Am. Phys, 55, 298, 1940.
2. Albright, F., Smith, P. H, and Richardson, A. M. Jour. Am. Med Assn, 116, 2465, 1941.
[illegible] M.: Rev. du Rheum, 3, 5, 1936
[illegible] 12, 407, 1952
[illegible]
6. Desmarais, M. [illegible]

7 Dobriner, Konrad Bull on Rheum Dis, 2, #3, Nov 1951.

1947

13 Kylin, E. Deutsch Arch f klin Med, 178, 217, 1935.

14 Marie, P. Rev de med, 6, 297, 1886

p 151, 1952

21. Staub, P L, Menthe, J W, Nelson, S S, and Cohn, H J Clin. Investig, 29, 349, 1950

22 Thomas, H M, Jr Arch Int Med, 51, 571, 1938

23 Turner, L W Personal Communication

24 Venning, E H, Johnson, L G, and Rose, B Proc. Second Clin ACTH Conf, Philadelphia, The Blakiston Co, 2, 49, 1951

pregnancy, a remission due to jaundice may persist for several months, and in exceptional instances for two or three years. In a few cases, the disease has recurred in a milder form.

The Mechanism of Action.—This, like that of pregnancy effect, is a mystery. The cholemia itself could be a factor, and this is suggested by the fact that the effect may occur early in obstructive jaundice when the liver is not believed to be damaged. Another possibility is that liver

of the rheumatic diseases. Also the damaged liver may fail to inactivate Compound F (hydrocortisone) and thus temporarily conserve it in the body.

or

de

liver function is disturbed at all, it could just as logically be increased as decreased An increased function would escape detection by our present tests

To date no single agent relating to the liver or bile constituents has been found to be responsible for the anti-rheumatic effect of jaundice.

ATTEMPTS TO REPRODUCE THE JAUNDICE PHENOMENON

Efforts at therapy by reproducing the jaundice effect fall into two main categories, first, the administration of bile products These include bile salts, both natural and synthetic, diluted ox bile and human bile, blood transfusions from jaundiced patients, and a combintaion of bile salts and bilirubin *None of these ingenious attempts has produced a satisfactory treatment* or indeed any serious claim to any anti-rheumatic effect

In the second category are attempts to reproduce the anti-rheumatic effect by hepatotoxic agents Synthetic compounds such as toluylene diamine and also lactophenin are capable of being administered in doses sufficient to cause a moderate hepatitis and jaundice with some anti-rheumatic effect but are potentially dangerous and have never come into practical use Good remissions have been produced by infecting rheumatoid patients with the secretions of patients with infectious hepatitis. MacCallum *et al* [12 23] produced hepatitis in about 10 per cent of inoculated volunteers and noted a satisfactory remission, averaging about 40 days, in 25 of 32 of these patients The method however cannot be recommended because of its inherent danger and because it can at best produce a temporary remission which probably could not be repeated on account of the acquired immunity which one "treatment" would confer

An anti-rheumatic effect has been claimed for heparin and is briefly mentioned here since it might have some relation, as yet unclarified, to the role of liver function in rheumatic disease Heparin acts as an anti-hyaluronidase, possibly by competing with the substrate since it is itself a mucopolysaccharide. (Specific antihyaluronidases are antibodies to the gamma globulin fraction of serum proteins) In general there is an increase in antihyaluronidases in several of the collagen diseases and in various liver diseases Dicoumarol, and Paritol-C (a polysulfuric acid ester of poly-

CHAPTER 29

THE AMELIORATING EFFECT OF PREGNANCY AND JAUNDICE ON CERTAIN RHEUMATIC DISEASES

By John Lansbury, M.D.

It is now well established that during pregnancy there may occur a suppression of the manifestations of a number of diseases in which hypersensitivity is believed to be an etiologic factor. This effect has been noted in migraine, asthma, angioneurotic edema, primary fibrositis, intermittent hydarthrosis, psoriatic arthritis, rheumatoid arthritis and rheumatoid spondylitis A similar suppressive effect in rheumatoid arthritis and several *others of the diseases listed may occur in the course of hepatitis and* jaundice As is now well known, these suppressive effects of both pregnancy and jaundice were the basis from which Hench successfully reasoned his way to the discovery of the anti-rheumatic action of cortisone.[19]

The naturally occurring suppressions of the rheumatic and allergic diseases are of importance because they suggest a new approach to the therapy of arthritis through the use of pregnancy products, and because a fuller understanding of the mechanisms involved may throw some light on the fundamental nature of rheumatic diseases

CLINICAL ASPECTS OF THE PREGNANCY EFFECT

In 20 of 22 cases of pregnancy occurring in young rheumatoid arthritics *a notable suppression of arthritic activity was reported by* Hench.[18] Luchessi[27] observed great amelioration in 14 of 18 pregnancies occurring in 8 patients, 6 of whom had peripheral rheumatoid arthritis and 2 rheumatoid spondylitis If relief has been attained in one pregnancy, subsequent pregnancies generally, but not always, cause similar relief. Occasionally arthritis may undergo an exacerbation during pregnancy, or may even have its onset during gestation. Similar variations in the effect of pregnancy on allergy are observed

Relief of arthritic pain may begin as early as the end of the first month of gestation but in some cases does not appear till the last trimester. In general the character of the relief is similar to that obtained from ACTH and cortisone with varying degrees of lessening of pain, swelling, and stiff- [illegible] increase in feeling of

[illegible]

rare instances persisting for several months The return of symptoms [illegible] be insidious or sudden and bears no constant relationship to the return of *menstruation* or the termination of lactation.

CHAPTER 30

DIET AND VITAMINS IN RHEUMATOID ARTHRITIS

By Edward F. Rosenberg, M.D.

Uncertainty concerning the cause of rheumatoid arthritis has been responsible for much controversy as to suitable diets for patients with this disease. Not infrequently, investigators in this field, having been persuaded to some special hypothesis as to etiology, have proposed a diet presumably fitting these hypothetical causes. Some such proposed diets gained widespread, though temporary, approval; but after months or years of trial, all have been discarded. It is today almost universally acknowledged that *rheumatoid arthritis cannot be overcome by any dietary manipulations which have thus far been proposed.*

Special therapeutic diets for rheumatoid arthritis in recent years have generally been based on speculations of rather illogical character. It has been proposed, for example, that autointoxication from intestinal fermentation and putrefaction is the cause.[4] To reduce or halt such undesirable intestinal reactions, it has been recommended that patients reinforce the diet with milk containing acidophilus organisms to change "intestinal flora." Some have sought to reduce hyperacidity by alkaline residue diets.[26] Some attempted to overcome the disease by removing foods to which the patient was presumably allergic.[29] One diet was aimed at dehydration to encourage removal of fluid from joints or other affected tissues.[22,25] Another aimed at overcoming constipation,[24] and still another diet high in protein was planned to overcome a supposed deficiency of protein in the blood.[23]

Not one of these diet plans has stood the test of time. One of the best known anti-rheumatoid arthritis diets, the low-carbohydrate diet, suggested by Pemberton[20,21] to correct a supposed defect of carbohydrate metabolism (*i.e.* a reduced carbohydrate tolerance) was employed widely throughout this country during recent decades but is now looked upon as useless.[3,11,23]

The majority of clinicians who treat rheumatoid arthritis today favor a generous intake of food, a diet containing adequate amounts of protein, carbohydrates, and fat, necessary amounts of minerals, and sufficient vitamins to prevent deficiencies.[3,4,8,11,15,23] It is worthy of note in this regard that in 1863, Sir A. B. Garrod, an English rheumatologist who devised the term "rheumatoid arthritis," had come to the conclusion that the only diet necessary for this condition was "as nourishing a diet as (the patient) is capable of properly digesting." This is a dictum, simply st[illegible]

in [illegible]

pa[illegible]

directions should be carefully transmitted in each instance in a manner

MECHANISM OF THE PREGNANCY EFFECT

This is at present a mystery. However sufficient research has been done to warrant a discussion of several possible mechanisms as noted below:

1. The Fetal Adrenals.—These are not developed until the 3rd month of gestation at which time the cortex is made up of a temporary structure,

unconfirmed) of injections of umbilical cord serum[33] does not prove that the effective factor is of fetal adrenal origin since the chorionic component of the placenta forms a large part of the fetal circulation.

2. The Corpus Luteum.—Progesterone from the corpus luteum is secreted very early in pregnancy and could therefore theoretically be a factor in the antirheumatic effect noted in the first few weeks of gestation Attempts to reproduce the pregnancy effect by intramuscular administration of progesterone[21,26,32,39] must on the whole be regarded as unsuccessful. Nevertheless there is some evidence of a suppressive effect even though this is erratic and unpredictable. One may conclude that, although pro-

3 Relaxin.—Relaxin is an ovarian hormone of unknown chemical composition. It is thought to be a protein. It is probably secreted by the

suggests itself.

Relaxin, prepared from sow's ovaries,* was administered by us in 1950 to one case of typical rheumatoid spondylitis and one case of typical peripheral rheumatoid arthritis The patients were "primed" by 5 mgm. of estrone intramuscularly for 3 days and then given relaxin, 1 mg. hypodermically every six hours for 9 successive days No change, beyond the expected variations of the disease, was observed.

4. The Placenta.—The placenta produces numerous hormones any one of which, or any group of which, might conceivably be the factor causing suppression of rheumatic activity Theoretically this action might be a direct one on the tissues of the mother, or it might be indirect through an influence on the mother's adrenal cortex, pituitary or other glands. Both adrenal cortex and anterior pituitary glands are known to undergo hypertrophy during pregnancy. Attempts to identify the anti-rheumatic placental factor are briefly noted below.

* Kindly supplied by Eli Lilly & Co

that will insure co-operation. Sufferers from rheumatoid arthritis include individuals of all age groups, children, mature adults, and the aged Some are overweight and a somewhat larger proportion have lost weight Some have health problems other than arthritis· allergies, diabetes mellitus, cardiac defects, peptic ulcers, constipation, etc Each of these additional or complicating defects will dictate its own requirements as to diet and will need proper consideration for planning food intake in these individuals

BASIC DIET FOR RHEUMATOID ARTHRITIS
(3,000 calories)

Dietary Pattern	Sample Menu	Approximate Measurf
Breakfast		
Fruit	Grapefruit	½ medium
Cereal	Whole grain cereal	½ cup (cooked)
Egg	Soft cooked egg	1
Bacon	Bacon	2 strips
Bread	Whole wheat toast	2 slices
Butter	Butter	2 squares
Beverage	Coffee	1 cup
Cream	Cream	½ cup
Sugar	Sugar	1 tablespoon
Jelly	Jelly	1 rounded teaspoon
Dinner		
Soup	Cream of potato soup	½ cup
Meat or subst	Roast beef and gravy	2 oz & ¼ cup
Potato or subst	Mashed potato	½ cup
Vegetable	Buttered new cabbage	½ cup
Sàlad	Sliced tomato	½ large
Salad dressing	Mayonnaise	1 tablespoon
Dessert	Vanilla ice cream	½ cup
Milk, ½ cream	Milk, ½ cream	½ pint
Bread	Whole wheat bread	2 slices
Butter	Butter	2 squares
Jelly	Jelly	1 rounded teaspoon
Supper		
Soup	Cream of pea soup	½ cup
Meat or subst.	Cold sliced ham	2 ounces
Potato or subst	Baked potato and butter	1 medium
Vegetable	Buttered green beans	½ cup
Salad	Celery & Carrot sticks	3–4 of each
Dessert	Chocolate pudding	½ cup
Milk, ½ cream	Milk, ½ cream	½ pint
Bread	Whole wheat bread	2 slices
Butter	Butter	2 squares
Jelly	Jelly	1 rounded teaspoon
Between-Meal Feeding		
Milk drink, ½ cream	Chocolate malted milk, ½ cream	

Immune Placental Globulins.*—Trial in a few cases of rheumatoid arthritis of immune placental globulins gave negative results in the author hands. A more extensive trial of this type of therapy is perhaps justified.

Placental Adrenocorticotropins.—Jailer and Knowlton[22] found adrenocorticotropic activity in acetone-dried human placentas which was not destroyed by boiling—a procedure which does destroy gonadotropic and posterior pituitary hormones. Tarantino[35] identified a corticotropic substance *in the chorion but not in the decidua of bovine placentas.* Opsahl, Long and Fry[30] demonstrated a hyaluronidase inhibiting substance in the urine of pregnant women which was apparently distinct from chorionic gonadotropin, and later, Opsahl and Long[31] obtained fractions from fresh placental tissue with corticotropic activity comparable to that of commercial preparations of ACTH.

Chorionic Gonadotropins.—Opsahl, Long and Fry[30] found corticotropic activity in chorionic gonadotropins prepared from the urine of pregnant women (presumed to be of placental origin). The activity remained after destroying the gonadotropic activity by heat thus indicating that corticotropic and gonadotropic factors are separate entities. It is therefore assumed that gonadotropins per se have no corticotropic action. Attempts to treat rheumatoid arthritis with gonadotropin, which will be discussed later, confirm their lack of anti-rheumatic activity.

Placental Steroids.—The status of progesterone has already been discussed under the heading of the Corpus Luteum. Salhanick *et al*[33] have recently isolated progesterone from the human placenta, thus confirming previous beliefs as to its presence there. Johnson and Haines[22a] have [illegible]

Commercial estrogens from various sources, placental or otherwise, have long since been discounted as effective anti-arthritic agents. Dobriner[11] noted a tremendous excretion in pregnancy of *pregnanolone* and *pregnanediol,* which are metabolites of progesterone, and a great increase in estrogens. Also he found a pronounced decrease in the excretion of *androsterone, 11-hydroxyandrosterone* and of *etiocholanolone.*

Wolfson, Robinson and Duff[40] find no proof that an increase of Compound F (hydroxycorticosterone) is the cause of the pregnancy effect. They point out that non-pregnant humans respond to an intradermal injection of human colostrum with a rapidly appearing wheal and flare. This reaction is abolished by pregnancy but was unaffected by full dosage of ACTH and cortisone in 12 non-pregnant subjects. They suggest that anti-allergic factors, distinct from an increase in glucocorticoids (Compounds E and F) may be responsible for the remissions of hypersensitivity disorders in pregnancy. Clarification of the possible role of glucocorticoids in the pregnancy effect must await studies of Compound F levels in blood and urine of pregnant women and a search for it in the placenta.

Altered Serum Constituents in Pregnancy.—The pregnant state is associated with widespread bodily changes. Among these, alterations in blood lipids, proteins and amino acids have been studied in a search for the mechanism whereby pregnancy suppresses rheumatic activity.

* Kindly supplied by Sharpe & Dohme

receiving suboptimal "non-toxic" doses of cortisone and noted no increased effectiveness of the cortisone.[16]

Vitamin B_{12} in Treatment of Arthritis.—Within recent months, symptomatic relief of osteoarthritis and osteoporosis were reported to follow *administration of vitamin B_{12} in large amounts.*[13] Crystalline vitamin B_{12} was given in weekly doses of 30 to 900 micrograms intramuscularly. Independent studies have not yet convincingly supported these reports. Use of this substance in treatment of three cases of rheumatoid arthritis was not successful.[12]

CONCLUSIONS

The diet for a patient with rheumatoid arthritis should be adequate in calories, protein, fat, and carbohydrate for the individual.[1,4,6,11,15,23] Vitamins should also be provided in adequate amounts. If the patient cannot *tolerate foods with sufficient content of the various vitamins, supplements* should be prescribed

[illegible]

have been shown to be a cause or even a consistent part of rheumatoid arthritis, nor has the administration of large doses of any vitamin been shown to produce a beneficial effect on the disease process.

BIBLIOGRAPHY

1 Abrams, N. R, and Bauer, W. Jour Am Med Assn, 111, 1632, 1938
2 Barrow, J V Med Jour and Rec, 136, 147, 1932
3 Bauer, W · Jour Am Med Assn, 104, 1, 1935
4 Boots, R H Internat Clin, 3, 154, 1935
5 Bywaters, E G L, Dixon, A St. J, and Wild, J B.· Lancet, 1, 951, 1950
6 Copeman, W S C.. Clin Jour, 63, 501, 1934
7. Copeman, W S C, *et al* Brit Med. Jour, 1, 1006, 1950.
[illegible] Therap, 16, 537, 1935
[illegible] our Med Sci, 179, 918, 1930
[illegible] 19, 1165, 1942.
[illegible] Med., 55, 403, 1935
[illegible], P. New England Jour Med, 223, 92, 1940
13 Hallahan, J D Am Pract and Dig of Treat, 3, 27, 1952
14 Hartfall, S J, and Harris, R.: Lancet, 2, 1202, 1949.
15 Hench, P. S · Nelson's Loose-Leaf Living Surgery, Thos Nelson and Sons, New York, p 104, 1935
16. ——— Internat. Rev of Vitamin Research, 23, 3, 1952
17. Kauffman, P., Beck, R D, and Wiseman, R D.: Jour. Am. Med Assn, 134, 688, 1947
18. LeVay, D., and Loxton, G E.: Lancet, 1, 209, 1950
19. Lewin, E, and Wassen, E. Lancet, 2, 993, 1949
20 Pemberton, R Am Jour Med Sci, 206, 498, 1943
[illegible] Arch Int Med, 25, 243, 1920.
[illegible], 653, 1934
[illegible] iseases (Reported by Hench,
[illegible] oc., 331, 1932
[illegible], 8, 1247, 1935
[illegible] 41.
29 Turnbull, J. A Jour Digest. Dis, 11, 185, 1944.

During gestation there is a marked increase in plasma neutral fat and later on in cholesterol and phospholipids. This occurs equally during [illegible]

cholesterol[22] Various serum lipids are increased during ACTH and cortisone administration. This elevation occurs gradually and is preceded by the anti-rheumatic effect. [illegible]

any, is at present unknown

During normal gestation there is a general lowering of total serum proteins due to steadily increasing hydremia; there is also a relative decrease in albumin, an increase of alpha and especially beta globulin but no [illegible]

poliomyelitis which could be related to its increased globulin level. The fibrinogen is increased despite the lowering of the total serum proteins [illegible]

We have no proof that it is related to changes in serum proteins, blood lipids, placental steroids, chorionic gonadotropins, or to ovarian progesterone or relaxin Placental corticotropins are perhaps the most promising mechanism so far suggested but much more investigation will be necessary before their role, if any, in the production of the pregnancy effect can be evaluated.

THERAPEUTIC ATTEMPTS WITH PREGNANCY PRODUCTS

Opinion as to the therapeutic value of pregnancy products depends upon what criteria are used in judging the therapy of rheumatoid arthritis. If we take as our criteria the following: (*1*) Improvement to be judged on [illegible]

* Through the courtesy of Eli Lilly & Co we administered an albumin fraction of post-partum pregnancy plasma to a small series of rheumatoid arthritics No real anti-rheumatic effect was observed

PART IV

IMPORTANT RHEUMATIC DISEASES CLOSELY ALLIED TO RHEUMATOID ARTHRITIS

CHAPTER 31

SPONDYLITIS

By EDWARD W. BOLAND, M.D.

SPONDYLITIS, in the broad sense, means arthritis of the spine The term [illegible]

pal structures of the spine The only true joints of the spine are the small posterior intervertebral (apophyseal) and the costovertebral articulations, these are diarthrodial or synovial joints The articulations formed by the vertebral bodies and the cartilaginous discs are devoid of synovial membrane and represent synchondroses. For practical purposes the sacro-iliac [illegible]

paraspinal muscles—must be thought of as spinal structures.

Probably more confusion exists in the clinical, pathologic and roentgenographic differentiation of spinal arthritis than in similar differentiation between types of arthritis involving peripheral joints Although often disagreeing as to subtypes, most authorities recognize two main divisions equivalent to the two common forms of peripheral arthritis; these are rheumatoid (ankylosing or atrophic) and osteoarthritic (hypertrophic or degenerative) spondylitis Special forms due to such specific causes as trauma, tuberculosis, pyogenic infections, brucellosis and syphilis are recognized also.

RHEUMATOID (ANKYLOSING) SPONDYLITIS

Rheumatoid spondylitis is a chronic and usually progressive disease of the [illegible]

reasonable and not too stringent, *we must immediately state that no successful treatment of arthritis by any pregnancy product has yet been established.* This however should not deter us from briefly reviewing several attempts in this promising field of investigation. Neither does this imply that interest- ... ut these

... ll to remember that any successful action of any pregnancy product in the future will probably be only a suppressive action. There is little reason to expect that the course of the disease would be permanently altered.* Furthermore, it is possible that a therapeutically successful pregnancy product would also cause some of the unwanted physiologic alterations which normally occur during pregnancy. The anti-rheumatic effect of pregnancy may be the result of several rather than one agent, in which case the use of one hormone alone might be expected to give only a partial effect.

...

However, this theoretical advantage would probably be nullified by the great difficulty of obtaining sufficient plasma products from maternal or cord blood to meet the needs of thousands of patients suffering from rheumatoid arthritis.

Barsi in 1947[4] claimed to have induced several long-lasting remissions in arthritis by repeated transfusions of blood from pregnant donors. Holbrook[20] found 2 or 3 dramatic results in a series of over 100 cases treated by this method but concluded that the *method is technically inapplicable to the thousands of patients with rheumatoid arthritis.*

Granirer and Victor, in 1949,[16,17] reported on a series of 8 cases of rheumatoid arthritis treated with post-partum pregnancy plasma, 250 cc. infusions once weekly for 3 to 6 months. Granirer[15] noted major improvement in 90 per cent of cases lasting from 3 weeks to 2 years after therapy was discontinued. Norcross[29a] observed *no benefit in 6 patients treated with 250 cc. infusions of post-partum pregnancy plasma at weekly intervals for 2½ months.*

Tufts *et al.*[38] in 1950 reported favorably on the use of pooled umbilical cord serum. Aronson *et al.*[2] in 1952 reported a series of 35 cases of arthritis treated by injections of umbilical cord serum, 10 cc twice weekly. A slight ...

ATTEMPTS AT TREATMENT WITH PREGNANCY HORMONES

Progesterone.—A trial of progesterone therapy in arthritis was first reported by Touw and Keipers in 1938.[37] Following this a total of 96 cases

* In 1931 the author treated a small series of cases of rheumatoid arthritis with acetone ...

pregnancy plasma

the disease calcific and osseous changes develop in the paraspinal ligaments, and when advanced these give rise to the well-known roentgenographic picture of a "bamboo spine." But if the present American concept is true, that rheumatoid spondylitis is merely the spinal variant of rheumatoid arthritis, the disease must be looked upon as a general systemic process in which the predominant manifestations occur in the spinal joints and their related structures.

Historical Sketch.—Descriptions and photographs of skeleton specimens in various museum collections give evidence that rheumatoid spondylitis has existed from the earliest times. Ruffer and Reitti described the skeleton of a man who lived during the third dynasty (2980 to 2900 B.C.) whose spinal column from the fourth cervical vertebra to the coccyx, and presumably in its total length, was diseased and transformed into a solid block because of new bone formation in the longitudinal ligaments. Other ligaments were involved also, but the vertebrae and the intervertebral spaces were unaffected. A skeleton found in Nordpfalz, dated (by tomb gifts enclosed) about 400 B.C., was reported by Arnold as showing similar [illegible]

rheumatoid spondylitis.

Probably the first acceptable description of the disease was made in 1691 by Bernard Connor, who wrote: "I have lately seen in France part of a [illegible] ... of the ilium, the os sacrum, the five vertebrae [illegible]

from one another were here so straightly and intimately joined, their ligaments perfectly bony and their articulations so effaced that they really [illegible] ... one bone, so that it was as easy to break one of [illegible] ... vertebrae, or [illegible]

any marks of them. [illegible] worthy that Connor c[illegible] associated with the malady: "From this construction of the parts, the body of this person must have been immovable, that he could neither bend nor [illegible] ... "and [illegible] , probably of [illegible] aden (1863), Blezinger (1864), Bradhurst (1866), Virchow (1869), Harrison (1870), Fl[illegible] ... [illegible] ise is credited to vo[illegible] ... ed to be a new neurologic disease characterized by stiffness of part or all of the spine, [illegible]

of various forms of arthritis, mostly rheumatoid arthritis or spondylitis, have been reported in the American and English literature[1,23 24 25 22 29] While there are differences in methods of scoring, the therapeutic claims are not as variable as would be expected. Of the 96 cases 11 are claimed to have a major clinical improvement. These apparently relapsed after [illegible] er day. [illegible] rate or [illegible] bleed-ing, somnolence and loss of hair were observed but no fluid retention was reported.

On the basis of the above studies, a capricious and unpredictable suppressive effect in about 10 per cent of cases of rheumatoid arthritis must be admitted. *However this in no way indicates that progesterone is a useful treatment for rheumatoid arthritis. None of the authors whose work is referred to advocate it as such. We must not confuse an interesting effect with a satisfactory treatment.*

Chorionic Gonadotropins.—Brown and Bradbury[3] in 1947 produced a condition of "pseudo-pregnancy" in normal women by the use of human [illegible]

a previous good spontaneous remission during pregnancy, with 5,000 to 10,000 international units of chorionic gonadotropin for 21 days No anti-rheumatic effect was observed The Ascheim-Zondek test remained negative

Dillon and Majnarich[10] in 1951 reported subjective improvement in 3 cases of rheumatoid arthritis with improvement in the sedimentation rate in 1 case, following injections of placental extract. This fraction was prepared by acetone extraction of human placenta which was further extracted by ethylene dichloride. It is possible that this may contain placental corticotropins, and it is hoped that this will be further studied

Estrogens.—It is generally agreed that estrogens of various types and from various sources are ineffective, in ordinary dosage, in rheumatoid arthritis. They may however be of use in various types of rheumatism arising at the time of the menopause, especially in cases of diffuse joint and muscle pains which would be classed as primary fibrositis. Whether [illegible]

action is available

A new claim by French rheumatologists[9] of successful treatment of rheumatoid spondylitis by high dosage of synthetic estrogens awaits confirmation in this country. Coste and Bonfils[9] treated a series of 25 male cases of ankylosing spondylitis by intramuscular injections of 20 mg. or more daily of diethylstilbestrol with later implantation of 300 mg. at intervals of 6 to 12 months. Sixteen of the 25 cases obtained varying degrees of subjective and objective improvement. Undesirable side effects were feminization with painful engorgement of the breasts and relative or total impotence.

Summary.—Attempts to reproduce the pregnancy effect in the management of arthritis by transfusions of pregnancy blood, post-partum pregnancy plasma, umbilical cord blood, progesterone, relaxin, immune placental globulins, chorionic gonadotropins, various estrogens and placental *corticotropins have so far failed to prove these agents as an established treatment of rheumatoid arthritis* despite the fact that a certain degree of antirheumatic activity is clearly demonstrated by several of them

ADVISABILITY OF PREGNANCY IN THE ARTHRITIC

This is a decision of the greatest importance which requires wise and mature clinical judgement in which the total life situation of the patient and her husband, their emotional needs, their financial resources and an accurate appraisal of the activity, extent and prognosis of the arthritis must all be carefully weighed

If the disease is active or far advanced so that the patient has difficulty in caring for herself and her house, obviously a pregnancy could be considered only if the patient has the full-time services of a nurse and a maid, or members of the family who can really be counted on to perform these functions. If the disease is mild with little functional impairment, preg- [illegible]

crease in total stress which often precipitates a serious and sometimes permanent worsening of the disease. In this type of case, pregnancy should only be advised if adequate assistance in the care of the home and the baby is guaranteed for at least 6 months after delivery, and preferably longer.

THE AMELIORATING EFFECT OF JAUNDICE

As already mentioned, spontaneous jaundice may cause a remission in rheumatoid arthritis and periarticular fibrositis. This effect was first carefully studied and its importance realized by Hench.[18] He also observed relief of pain in psoriatic arthritis, osteoarthritis and intrapelvic protrusion of the hips, but the pain of gout, neuralgia and neuritis was not benefited, perhaps because the degree of jaundice was too slight. (Remissions *in hay fever and migraine have also occurred during spontaneous* jaundice but these effects have not been intensively studied.) It would thus seem that the jaundice effect is of a general nature and not specific for rheumatoid arthritis. *Almost any kind of obstructive* or intrahepatic jaundice can produce the effect, but hemolytic jaundice apparently does not do so.

The anti-rheumatic action of jaundice appears to be constant if the jaundice is sufficient to cause a serum bilirubin of 8 to 10 mg per cent, although milder degrees may also produce the effect.

Character of the Remission.—Suppression of arthritic manifestations usually occurs by the time jaundice is noted and possibly earlier. The duration of the remission is not directly related to the depth and duration ndice in all cases. In contrast to the rapid relapse of arthritis after

bulk of American investigators favor the concept that the disease is a spinal variant of rheumatoid arthritis.

Incidence.—Contrary to general belief, rheumatoid spondylitis is a common cause of back disability, especially in young men. Large series have been reported, ranging from 100 to 1035.[41] In civilian series approximately one case of rheumatoid spondylitis has been observed to every thirteen or nineteen cases of rheumatoid arthritis involving peripheral joints.[10,24,57] Because the disease has a predilection for young men of military age it was a common cause of disability among military personnel during World War II. Eighteen per cent of all soldiers with chronic backache admitted to one army general hospital had the disease.[4] At a United States Army Rheumatism Center, 1084 cases of spondylitis were admitted during a period of two years.[7] These comprised 18.1 per cent of 6000 consecutive admissions for all types of rheumatic disease, almost as many soldiers being admitted for spondylitis as for peripheral rheumatoid arthritis. At one British installation the disease made up 5.2 per cent of admissions for rheumatic complaints.[42]

Rheumatoid spondylitis most commonly begins between the ages of sixteen and forty years. In one series of a hundred cases 3 began in the second decade, 44 in the third decade, 32 in the fourth decade, 13 in the fifth decade, and 6 in the sixth decade; in the remaining cases the age at onset could not be definitely determined.[13] In another series of 100 cases the [illegible] years [illegible] en the

[illegible] relative sex incidence favoring males varies in different series from 4:1 to 20:1.[10,12,57]

[illegible] in rheuma- [illegible] heredity: [illegible] tients with [illegible] heumatoid [illegible] thirty times more prevalent [illegible] tigation by [illegible] 9 per cent [illegible] that this

studied by Boland and Present.[7] [illegible] mediate precipitating or predisposing factor could be elicited, and in the remaining 20 per cent no constant factor was found. In 8 per cent the first [illegible] more

was prevalent in older papers. Marie[16] and others [illegible] could cause this disease, and Forestier[14] believed the primary focus to be in the genito-urinary tract with the toxic products reaching the pelvis and

pregnancy, a remission due to jaundice may persist for several months, and in exceptional instances for two or three years In a few cases, the disease has recurred in a milder form.

The Mechanism of Action.—This, like that of pregnancy effect, is a mystery. The cholemia itself could be a factor, and this is suggested by the fact that the effect may occur early in obstructive jaundice when the liver is not believed to be damaged Another possibility is that liver

of the rheumatic diseases Also the damaged liver may fail to inactivate Compound F (hydrocortisone) and thus temporarily conserve it in the body.

or

dc

liver function is disturbed at all, it could just as logically be increased as decreased An increased function would escape detection by our present tests

To date no single agent relating to the liver or bile constituents has been found to be responsible for the anti-rheumatic effect of jaundice

ATTEMPTS TO REPRODUCE THE JAUNDICE PHENOMENON

Efforts at therapy by reproducing the jaundice effect fall into two main categories, first, the administration of bile products These include bile salts, both natural and synthetic, diluted ox bile and human bile, blood transfusions from jaundiced patients, and a combintaion of bile salts and bilirubin. *None of these ingenious attempts has produced a satisfactory treatment* or indeed any serious claim to any anti-rheumatic effect.

In the second category are attempts to reproduce the anti-rheumatic effect by hepatotoxic agents Synthetic compounds such as toluylene diamine and also lactophenin are capable of being administered in doses sufficient to cause a moderate hepatitis and jaundice with some anti-rheumatic effect but are potentially dangerous and have never come into practical use Good remissions have been produced by infecting rheumatoid patients with the secretions of patients with infectious hepatitis MacCallum *et al*[12,23] produced hepatitis in about 10 per cent of inoculated volunteers and noted a satisfactory remission, averaging about 40 days, in 25 of 32 of these patients The method however cannot be recommended

tempo-

the ac-

An anti-rheumatic effect has been claimed for heparin and is briefly mentioned here since it might have some relation, as yet unclarified, to the role of liver function in rheumatic disease Heparin acts as an anti-hyaluronidase, possibly by competing with the substrate since it is itself a mucopolysaccharide. (Specific antihyaluronidases are antibodies to the gamma globulin fraction of serum proteins) In general there is an increase in antihyaluronidases in several of the collagen diseases and in various liver diseases Dicoumarol, and Paritol-C (a polysulfuric acid ester of poly-

Clinical Picture.—**Mode of Onset.**—Because the real diagnosis is often overlooked for years the clinical features which characterize the onset are of considerable importance The early symptoms of rheumatoid spondylitis are frequently mislabelled as muscular rheumatism, fibrositis, lumbago, chronic low back strain, idiopathic sciatica or even kidney disease The onset is insidious in approximately 80 per cent of cases. Almost invariably the first complaints are referred to the lower back and consist of episodes of aching and stiffness, transient back pain or sciatica

Attacks of aching and stiffness, lasting a few days at a time and then subsiding completely or almost completely, are the most frequent initial complaints Sometimes these symptoms are persistent from the onset.

damp weather, and are temporarily relieved by local heat and by acetylsalicylic acid Although the intensity varies, the early symptoms are mild in most cases In a few, disabling attacks often described as "lumbago" occur Later, the aching and stiffness tend to be more persistent and to lose their typical "fibrositis" characteristics

Less often the first symptoms consist of transient sharp pains or catches ... These may be precipitated ... ing, but at times they occur ... ness accompany such pains.

hours, the patient may be awakened about three or four o'clock in the morning and lies in discomfort unless he takes a hot bath or gets up and moves around

In about 10 per cent of cases the first symptom is sciatic pain This is ...

simulating visceral disease, are the initial symptoms. Sometimes progressive limitation of back motion without actual discomfort is the only complaint

13 per cent of cases[4]

So-Called Prespondylitic Stage.—Scott[45,46,47] and others[22] described a "prespondylitic stage," characterized by wandering pains in the shoulders, arms, ribs and legs during which the sacro-iliac joints presumably were silently involved They maintained that spinal symptoms do not develop until bony ankylosis of the sacro-iliac articulations is nearly complete There appears to be little evidence to justify the concept of a so-called

Summary.—Attempts to reproduce the pregnancy effect in the management of arthritis by transfusions of pregnancy blood, post-partum pregnancy plasma, umbilical cord blood, progesterone, relaxin, immune placental globulins, chorionic gonadotropins, various estrogens and placental corticotropins have so far *failed to prove these agents as an established treatment of rheumatoid arthritis* despite the fact that a certain degree of antirheumatic activity is clearly demonstrated by several of them.

ADVISABILITY OF PREGNANCY IN THE ARTHRITIC

This is a decision of the greatest importance which requires wise and mature clinical judgement in which the total life situation of the patient and her husband, their emotional needs, their financial resources and an accurate appraisal of the activity, extent and prognosis of the arthritis must all be carefully weighed.

If the disease is active or far advanced so that the patient has difficulty in caring for herself and her house, obviously a pregnancy could be considered only if the patient has the full-time services of a nurse and a maid, or members of the family who can really be counted on to perform these functions If the disease is mild with little functional impairment, pregnancy may be advised since, of itself, it seldom aggravates arthritis. The main problem is, then, the post-partum relapse which occurs at a time when the care of the baby plus the regular household duties constitute an increase in total stress which often precipitates a serious and sometimes permanent worsening of the disease. In this type of case, pregnancy should only be advised if adequate assistance in the care of the home and the baby is guaranteed for at least 6 months after delivery, and preferably longer.

THE AMELIORATING EFFECT OF JAUNDICE

As already mentioned, spontaneous jaundice may cause a remission in rheumatoid arthritis and periarticular fibrositis. This effect was first carefully studied and its importance realized by Hench.[18] He also observed relief of pain in psoriatic arthritis, osteoarthritis and intrapelvic protrusion of the hips, but the pain of gout, neuralgia and neuritis was not benefited, perhaps because the degree of jaundice was too slight (Remissions in hay fever and migraine have also occurred during spontaneous jaundice but these effects have not been intensively studied.) It would thus seem that the jaundice effect is of a general nature and not specific for rheumatoid arthritis. Almost any kind of obstructive or intrahepatic jaundice can produce the effect, but hemolytic jaundice apparently does not do so.

The anti-rheumatic action of jaundice appears to be constant if the jaundice is sufficient to cause a serum bilirubin of 8 to 10 mg. per cent, although milder degrees may also produce the effect.

Character of the Remission.—Suppression of arthritic manifestations usually occurs by the time jaundice is noted and possibly earlier. The duration of the remission is not directly related to the depth and duration of jaundice in all cases. In contrast to the rapid relapse of arthritis after

becomes filled with granulation tissue, and later fibrous tissue adhesion
produce a fibrous synostosis. Simultaneously the juxta-articular bon
undergoes changes which from radiographic evidence consist of both des
truction and sclerosis. The articular surfaces appear roughened, chewe
up or moth eaten; the joint space becomes irregular, being wide at on
point and narrow at another. Areas of spotty and generalized osteoporosi
appear in the subchondral regions. The spotty areas evidently are due t
focal collections of round cells and later to granulation tissue, while th

becomes progressively more narrowed, and the ultimate picture is that o
bony ankylosis with disappearance of all vestiges of the joint.

Calcification and ossification of various *spinal ligaments (anterior, inter*

ossification remains a subject of controversy. Some contend that it repre
sents a reparative process secondary to loss of normal articular support o

and muscles are sites of inflammation in rheumatoid spondylitis, and in

and even extension of the spongiosa of the vertebrae through the central
part of the discs have been reported;[33] in these cases the involved spine may
structurally resemble a long bone. Such findings, however, represent an
extreme and uncommon change Usually the intervertebral spaces appear
normal roentgenographically even in advanced cases, and the vertebral
bodies usually remain uninvolved except for secondary bone atrophy. In
contrast to osteoarthritis (hypertrophic) of the spine, exostoses are un-
common

Involvement of the sacro-iliac joints is an important part of rheumatoid
spondylitis. In the vast majority of instances, but not in all, these joints

years may elapse before clinical manifestations or *x*-ray alterations appe...
higher in the spine In a relatively small percentage of cases the disease
may begin in the thoracic or lumbar region; this is more common in females.

Peripheral rheumatoid arthritis coexists in approximately 20 per cent of
cases. Biopsies taken from these joints show histopathologic changes
identical to those found in rheumatoid arthritis unassociated with spondyl-
itis.[41] Formerly, the shoulders and hips were considered the most common
sites of coexisting arthritis. In a recent investigation,[4] *however, other*
peripheral joints were more often involved The order of frequency was as
follows: knees, metatarsophalangeal, metacarpophalangeal, wrists, proxi-
mal interphalangeal (fingers or toes), hips, shoulders and elbows.

Clinical Picture.—Mode of Onset—Because the real diagnosis is often overlooked for years the clinical features which characterize the onset are of considerable importance The early symptoms of rheumatoid spondylitis are frequently mislabelled as muscular rheumatism, fibrositis, lumbago, chronic low back strain, idiopathic sciatica or even kidney disease. The onset is insidious in approximately 80 per cent of cases. Almost invariably the first complaints are referred to the lower back and consist of episodes of aching and stiffness, transient back pain or sciatica

Attacks of aching and stiffness, lasting a few days at a time and then subsiding completely or almost completely, are the most frequent initial complaints Sometimes these symptoms are persistent from the onset. The aching and stiffness present qualitative characteristics of "fibrositis," that is, they are most pronounced in the morning on arising, are accentuated by physical inactivity and ameliorated by mild exercise, are made worse by damp weather, and are temporarily relieved by local heat and by acetylsalicylic acid Although the intensity varies the early symptoms are mild
in most
occur.
their ty

Less often the first symptoms consist of transient sharp pains or catches
...itated
... occur
pains.

As the disease progresses the pain tends to become a constant dull discomfort, sometimes described as a "deep ache," a "tired feeling" or a "soreness," usually aggravated by activity which entails weight-bearing or movement of the back. Commonly the discomfort is worse during the early morning hours, the patient may be awakened about three or four o'clock in the morning and lies in discomfort unless he takes a hot bath or gets up and moves around.

In about 10 per cent of cases the first symptom is ...

plaint

General constitutional symptoms such as fatigue, weight loss, anorexia and low grade fever may accompany the onset Their intensity varies with ...

13 per cent of cases[4]

So-Called Prespondylitic Stage—Scott[45,46,47] and others[22] described a "prespondylitic stage," characterized by wandering pains in the shoulders, arms, ribs and legs during which the sacro-iliac joints presumably were silently involved. They maintained that spinal symptoms do not develop until bony ankylosis of the sacro-iliac articulations is nearly complete There appears to be little evidence to justify the concept of a so-called

teristic Later, in more severe cases, erosion, scalloping or irregularity of the joint margins may be apparent. As in the sacro-iliac joints the final event consists of bony ankylosis, but even at this stage all joints of an involved segment may not be fused

Changes in the Paraspinal Ligaments.—Calcification and ossification of the paravertebral ligaments usually do not develop until late in the disease. The anterior, lateral and posterior longitudinal ligaments and the ligamenta flava are the common sites of involvement. Most often calcification occurs first in the lower thoracic and upper lumbar regions All degrees of change



FIG 192 —Completely rigid cervical spine with ligamentous calcification and ankylosis of the posterior intervertebral (apophyseal) joints Note the absence of cervical lordosis

191 and 192)

***Clinical Course.**—This is extremely variable and to a large extent* is dependent on the severity of the disease[4] Mild cases, characterized by insidious onsets, little or no elevation of erythrocyte sedimentation rates, minimal or no constitutional symptoms and x-ray changes showing predominant juxta-articular sclerosis rather than spotty osteoporosis or joint mottling, tend to run a relatively benign course. Progression is gradual and the stage of "poker back" deformity may not be reached for fifteen to twenty-five years, but the *disease may subside temporarily or permanently at any point.* For example, it may terminate before it has extended beyond the

prespondylitic stage. In approximately one out of ten patients a history of migratory peripheral joint muscle complaints, with or without stiffness, may be elicited. Such complaints usually are not impressive. They often have characteristics which suggest an intramuscular and periarticular "fibrositis," and more correctly should be considered as a manifestation secondary to rheumatoid arthritis of the spine. With the exception of those patients having a preceding rheumatoid arthritis involving peripheral joints, the first definite rheumatic complaints are almost always located in the back



FIG 181 —Contour of a normal back When the disease is confined to the sacro-iliac joints the back contour may be normal and physical signs lacking (Boland and Present, courtesy of Jour Am Med. Assn)

Symptoms and Findings.—The overall pattern of rheumatoid spondylitis is fairly characteristic, but the clinical picture at the time of examination is

The disease may be mild, moderate or severe. It may be localized in the sacro-iliac joints, or at the other extreme all the synovial joints of the spine may be involved. One area may be semidormant and silent and another

sacro-iliac joints; hence the only residue may consist of the x-ray "ashes" of completely ankylosed sacro-iliac joints. However, the disease may become reactivated at any time even though it apparently has been burned out for years.

In cases of moderate severity, the disability is generally of sufficient degree to cause the patient to seek medical aid early in the course of the disease. Progression is more rapid, the sedimentation rate is almost invariably elevated, and the roentgenograms reveal changes which signify a higher degree of disease activity. Restriction of back motion, diminished chest expansion, lumbodorsal stoop and neck involvement may all be present within a period of ten years. The disease activity may fluctuate, however, and there may be alternating periods of temporary improvement and exacerbation

When the disease is severe, back disability and general constitutional reaction are pronounced and extension is rapid. Extreme deformity with complete rigidity of all segments of the spine may develop within three years after the onset.

Rheumatoid Spondylitis Should be Suspected

(1) When a young man complains of chronic recurrent or persistent low back aching and stiffness, especially when the erythrocyte sedimentation rate is elevated
(2) When a young man complains of persistent vague symptoms, such as a tired feeling in the lower back, low back soreness, catching pains in the lower back, buttocks or hips, especially when these symptoms are accompanied by an elevated erythrocyte sedimentation rate or general systemic symptoms
(3) When a young man complains of recurrent sciatic pain, particularly if it alternates from side to side and is accompanied by aching and stiffness of the lower back
(4) When persistent back symptoms develop in a patient with peripheral rheumatoid arthritis
(5) When a patient complains of thoracic girdle pains, especially if accompanied by such symptoms as aching, stiffness and restriction of motion in the back or limitation of chest expansion.
(6) When characteristic clinical signs and symptoms are present despite the absence of roentgenographic changes in the sacro-iliac and/or apophyseal joints, *several months or even years may elapse before diagnostic roentgenographic changes are demonstrable*
(7) When there is roentgenographic evidence of bilateral sacro-iliitis or characteristic x-ray abnormalities in the apophyseal joints

Diagnosis.—Diagnosis is not difficult when the disease has progressed sufficiently to present characteristic physical and roentgenographic signs. Adequate clues are furnished by such clinical features as *progressive limitation of spinal motion, persistent paravertebral muscle spasm, straightening of the lumbar spine, dorsolumbar kyphosis, restricted chest expansion, lumbar muscle atrophy, flattened anterior chest, protrusion of the neck,* etc. Roentgenographic alterations, such as *bilateral sacro-iliitis, apophyseal joint changes, straightening of the lumbar spine* and *calcification of paravertebral*

area active and symptomatic The severity of symptoms and the extent of

it
po-
ird
the
sacro-iliac joints are involved at the time of examination and, as a rule, the lumbar spine is affected before symptoms appear in the thoracic region

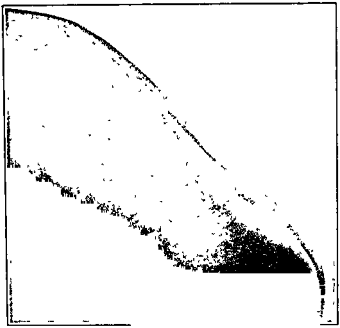


Fig 182 —Rheumatoid spondylitis involving the sacro-iliac joints and the lumbar spine Note the straightening of the lumbar spine and the "ironed out" appearance of the lower back

Sometimes, however, the disease may by-pass one or even two segments, consequently different combinations of symptoms and findings are encountered It would be impractical to describe all possible variations but an enumeration of the features resulting from involvement in individual segments may be useful.[4]

Sacro-iliac Involvement—With involvement of these joints alone the symptoms consist of aching, stiffness and pain in the lower back Sciatica is present in approximately 10 per cent of instances Tenderness over one or both sacro-iliac joints is present on percussion or deep palpation in about 50 per cent of patients; when ankylosis develops, tenderness and pain disappear. Orthopedic maneuvers which produce sacro-iliac joint movement may cause local pain Mild paravertebral muscle spasm without restriction of lumbar motion is frequent; when muscle spasm is pronounced it usually indicates lumbar apophyseal joint involvement. In mild cases or when the

ligaments serve to confirm the diagnosis Additional evidence is supplied by an *elevated erythrocyte sedimentation rate, general systemic symptoms or coexisting peripheral rheumatoid arthritis.* The spondylitic derelict with

which are observed before roentgenographic changes have developed Certain points summarized axiomatically may be of aid in the early recognition of the disease

Osteoarthritis of the spine (hypertrophic spondylitis—degenerative joint disease of the spine) is the chief disease from which rheumatoid spondylitis must be distinguished However, this disturbance seldom produces symptoms before the fifth decade, the sedimentation rate is normal, anemia,

of the jc
tion of
vertebra
bony bridges

Osteochondritis juvenilis dorsi (adolescent kyphosis) must be differentiated at times.[8] In this condition the process is restricted to the dorsal and

longitudinal ligament may occur, but in general the x-ray picture does not resemble that of rheumatoid spondylitis and the *sacro-iliac joints are not involved*

Sacro-iliac epiphysitis in adolescents may be confused with rheumatoid spondylitis. Similar symptoms are encountered including low back pain and stiffness, tenderness over the sacro-iliac joints and sciatic pain Roent-

measures

Because during the earlier stages of the disease sciatic pain is not uncommon, rheumatoid spondylitis must be differentiated at times from other causes of chronic low back disability accompanied by sciatica, especially from cases of ruptured intervertebral disc Occasionally the onset of spondylitis is dated to a back injury, and in early cases when physical or roentgenographic signs are minimal or absent differentiation may be difficult Increases in the protein content of the cerebrospinal fluid may occur in both spondylitis and ruptured intervertebral discs, thus spinal fluid examinations are of little differentiating value *However, the sciatica in spondylitis usually is mild, often alternates from side to side, and is unaccompanied by neurologic signs such as sensory or reflex changes* Spino-

sacro-iliac joints are ankylosed (and unaccompanied by higher involvement) physical signs may be absent entirely. (Fig. 181)

Lumbar Involvement.—This segment is rarely involved alone as almost invariably the disease has extended upward from the sacro-iliac joints. Pain, aching and stiffness as already noted remain the dominant symptoms; but they are not of great localizing value. Among the signs which signify involvement of the lumbar spine the following may be included: (1) limitation of motion of the lumbar spine, (2) paravertebral muscle spasm, (3) straightening of the lumbar spine, (4) tenderness to percussion and deep



FIG. 183.—Rheumatoid spondylitis showing limitation of lumbar motion and paravertebral muscle spasm (Boland and Present, courtesy Jour. Am Med. Assn.)

palpation over the lumbar spine, (5) pain on attempted forced flexion and extension of the lumbar spine, (
portion of the lumbar segment,
of the lumbar spine gives the lo
(7) anterior stoop (usually associated with thoracic involvement). (Figs. 182 and 183)

Thoracic Involvement.—When the thoracic segment is affected the same

chest expansion, (4) flattening of the anterior chest (expiratory position), and (5) thoracolumbar kyphosis (Figs 182 and 183)

alterations and its characteristic roentgenographic changes in the sacroiliac and apophyseal joints or in the paravertebral ligaments, differential diagnosis is easily made.

Treatment.—There is no known cure for rheumatoid spondylitis, but much can be accomplished towards ameliorating the symptoms and not infrequently complete arrest of the disease may reward therapeutic effort. If treatment is begun in the early stages, spinal deformity may be prevented or corrected.

Education of the Patient.—As with all victims of chronic disease, cooperation is best obtained by familiarizing the patient with the facts relating to the disability.[3] The patient should be fully instructed regarding the nature of the disease, its potentially crippling end results (especially if treatment is neglected), and what may and may not be expected from treatment If so indoctrinated, he is more likely to persist in carrying out the various exercises and home measures which form an essential part of a spondylitic regimen

General Measures.—*Because rheumatoid spondylitis is a systemic disease,* measures designed to improve general health are equally as important as in peripheral rheumatoid arthritis These should include a *high caloric diet* ... *salts* or ... Physi- ... l. With disease of mild or moderate severity at least ten hours of bed rest at night ...

physiotherapeutic measures often give symptomatic relief; occasionally, however, they seem to *aggravate* the symptoms, *particularly diathermy used during acute exacerbations of the process*

Prevention and Correction of Postural Deformities.—*Relatively simple exercises if carried out conscientiously day after day for a period of years do much to prevent postural deformity* The patient must be made to understand that ...

on making the patient posture conscious He should be taught to assume a proper stance at all times with the lower abdominal muscles pulled in, the ... head held erect In addition, ... pine ... ises ... l be accomplished at least twice daily at home. Simple analgesics, such as ... and/or a hot tub bath ... The spondyl- ... ls), but *without* ... hyperextension

Such instructions may be carried out as preventive measures even though postural deviations are minimal or have not yet developed

Cervical Involvement.—With involvement of the cervical spine the same general features are present as in other regions. The neck often assumes a protruded position. Motion in all directions may be markedly impaired and the patient may have to pivot his whole body in order to look sideward. Cervical muscle spasm may be pronounced and later atrophy of the neck muscles may ensue Cervical radicular pain is rare.



FIG. 184

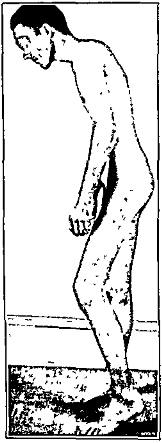


FIG 185

FIG 184 —Rheumatoid spondylitis involving all segments of the spine Note the flattened anterior chest, the pot belly and the forward protrusion of the head (Boland, courtesy Calif Med)

FIG. 185.—Typical spondylitic derelict with fixed flexion deformity of left hip (Boland and Present, courtesy of Jour. Am Med Assn)

General Symptoms.—These may be minimal or absent in mild cases; many patients are able to pursue useful occupations for years without appreciable alteration in general health. In severe or moderately severe cases, on the other hand, weakness or weight loss may be sufficiently pronounced to raise a suspicion of malignancy, tuberculosis or some other debilitating disease

In the majority of patients with early postural deformities correction may be accomplished by postural exercises alone. *Spinal braces* are usually reserved for those cases with more advanced disease and with postures which cannot be corrected or maintained by exercise alone. Sometimes spinal braces are employed when pain and muscle spasm cannot be relieved by other means Hyperextension frames, hyperextension beds and plaster half-shells are indicated only when spinal deformity cannot be corrected by other methods; occasionally plaster half-shells are needed to control muscle spasm and pain.

Roentgen Therapy.—*X*-ray therapy applied locally to the sacro-iliac joints and to all involved regions of the spine appears to be of value in the treatment of rheumatoid spondylitis [27,52,53] The immediate effects consist in reduction of pain, local tenderness and subjective stiffness; less often other manifestations, such as limitation of motion, restriction of chest expansion and general constitutional symptoms, are also improved The overall beneficial effects seemingly do not depend upon how severe the disease is or how much of the spine is affected. But the degree of improve- [illegible]

[illegible]

physical manifestations rarely occurs, more often improvement is partial The duration of beneficial effects from roentgen therapy cannot yet be appraised because, unfortunately, five and ten year controlled follow-up studies are not yet available. (See also chapter on Radiology in Arthritis, p 106.)

[illegible]

[illegible]

erythrocyte sedimentation rate returned to or toward normal, suggesting that in some the disease may have been arrested. Hare[24] and others[25,26,29,50] have reported similar results

The technique of therapy now used most commonly in the United States was described by Smyth, Freyberg and Lampe as follows Two hundred [illegible]

course Three courses of treatment, given at intervals of three to six [illegible]

the site of recurrent symptoms.

In general, the degree of constitutional reaction parallels the height of the erythrocyte sedimentation rate, but it is seldom pronounced in the ordinary case unless peripheral joints are involved also. Fever is usually absent or it is slight; it rarely exceeds 100° F. A tachycardia (90 to 100) is not uncommon. Iritis or iridocyclitis occurs with approximately the same frequency as in cases of peripheral rheumatoid arthritis.

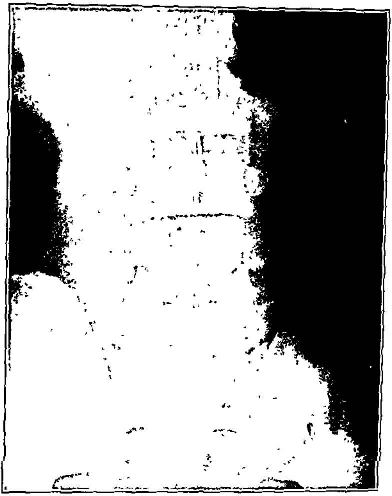


FIG. 186.—Normal sacro-iliac joints. Note the well-defined articular margins and the clear joint spaces. (Boland and Shebesta, courtesy of Radiology.)

Laboratory Findings.—The only laboratory test of value in rheumatoid spondylitis is the erythrocyte sedimentation rate. It serves as a fairly consistent gauge of disease activity. It is elevated in approximately 80 per cent of cases, the usual range being between 20 and 60 mm in one hour (Westergren method, normal under 16 or 20 mm in one hour). In mild but active cases the sedimentation rate may be within normal limits. A moderate degree of hypochromic anemia is present in about 30 per cent of cases.

That the results from roentgen therapy are not psychic in origin have been demonstrated by Smyth, Freyberg and Lampe,[52] and subsequently by a controlled study done on soldiers [50] In the latter study, 75 soldiers with rheumatoid spondylitis were divided into 3 groups of 25 each. One group was given roentgen therapy to all areas of the spine clinically involved The second group was given placebo therapy only, that is, the patients were brought to the x-ray department and went through the same routine as those receiving therapy, but actually they received no irradiation. The third group was treated with breathing and postural exercises and general symptomatic measures only The results from this study seemed to prove that the effects from roentgen therapy were not psychic in origin. Sixty-eight per cent of those receiving roentgen therapy obtained objective improvement and 92 per cent subjective improvement. In the placebo or psychotherapeutic group only 8 per cent showed objective improvement and 28 per cent subjective improvement. Those patients receiving only breathing and postural exercises had results intermediary between the two.

Whether roentgen therapy modifies the activity of the disease or merely produces analgesia resulting in a lessening of the clinical manifestations cannot yet be determined. Controlled follow-up studies are needed to clarify this important point Nevertheless, x-ray treatment applied to those areas of the spine actively involved with the disease serves as an important adjunct to treatment. Even if such treatment should prove to have only an analgesic effect it is worthwhile because it allows better results to be obtained from breathing and postural exercises. Roentgen therapy should not be relied upon as the only measure of treatment; it should be combined with other measures such as those designed to improve general health and to prevent or correct postural deformity.

Ill-effects from Roentgen Therapy in Rheumatoid Spondylitis.—It is not uncommon to find transient decreases in the number of leucocytes following roentgen therapy to the spine, but significant leucopenia is not seen commonly Even if marked leucopenia results, the leucocyte count usually returns to normal within three or four weeks if roentgen therapy is stopped. A reduction in dosage lessens the tendency toward leucopenia

Anorexia, nausea and vomiting may occur when the thoracic and lumbar regions are irradiated. Such symptoms may begin from four to six hours after therapy and usually subside in two to three days. When larger amounts of irradiation are used, these symptoms are usually more severe.

If it is necessary to treat the cervical spine, the patient must be warned that temporary depilation may result in the area treated. Sometimes it is more prudent, in women especially, to avoid irradiation above the hair-

[illegible] e doses [illegible] ed that [illegible] c joints

Cortisone Therapy.—As in rheumatoid arthritis involving peripheral [illegible]

sacro-iliac, apophyseal, and costovertebral [illegible] muscles. Manifestations resulting from structural changes in the joints or calcifications in the paraspinal ligaments are not altered. Thus muscle

The cerebrospinal fluid proteins may be elevated in rheumatoid spondylitis.[35] In a recent study they were elevated in 42 per cent of cases[6] The elevation is usually moderate in amount, ranging between 46 and 105 mg per cent *The protein content of the cerebrospinal fluid apparently bears* no consistent relationship to the duration of the disease or to the degree of spinal extension, but it seems to be related to the severity of the process

gold reaction, total cell count, manometric pressure and sugar content, are usually within normal range



Fig. 187 —Bilateral sacro-iliac involvement. Note the juxta-articular sclerosis and poorly defined joint spaces with mottling

Roentgenographic Findings.—The x-ray findings in rheumatoid spondyl-

aching and stiffness, muscle spasm, radicular pain and general debility may be benefited rapidly and markedly. Limited spinal motion or restricted chest expansion due to recent active involvement of the apophyseal or costovertebral articulations may be improved during cortisone therapy but fixed spinal deformities are not corrected. The therapeutic result achieved is

matoid spondylitis is a chronic and often progressive malady, the drug must be given more or less continuously for an indefinite period of time if improvement from it is to be sustained. As in peripheral rheumatoid arthritis, endocrine complications occur not infrequently with prolonged uninterrupted therapy (*see* Chapter 17). At the present state of our knowledge it seems advisable to employ cortisone as treatment of second choice for rheumatoid spondylitis, reserving it for those patients who fail to respond adequately to roentgen therapy and general measures.

The dosage plan followed is similar to that used for peripheral rheumatoid arthritis (*see* Chapter 17). Results are best when large suppressive amounts of the hormone are given at the beginning of treatment and the *dose then gradually reduced until the smallest dose needed to control the* symptoms is reached.[59 60 61] The general plan currently used consists of three stages. (1) period of initial large suppressive doses, (2) period of *gradual dose reduction; and (3) period of extended smaller maintenance*

with the initial activity of the process, being longer in cases of greater severity When satisfactory improvement is obtained, the dosage is then gradually reduced in step-like fashion, by 10 or 12.5 mg at a time and at intervals of every five to seven days The smallest dose reached which will uphold an adequate degree of improvement (not complete inactivation of the disease) is considered as the patient's maintenance dose, the size of which varies in individual cases and is dependent largely on the initial severity of the disease. Treatment is then continued without interruption for extended periods of time, with adjustments in the maintenance dose being made periodically as changes in disease activity demand (For

by injection (*see* Chapter 17)

await confirmation before this treatment is advised *Intravenous* [illegible]

cartilage or bone is modified sufficiently to be recorded on roentgenograms. Thus, the disease may be present for long periods before roentgenographic diagnosis is possible. Similarly, in patients with ascending disease there

located in the juxta-articular portion of the ilium, particularly at the caudal

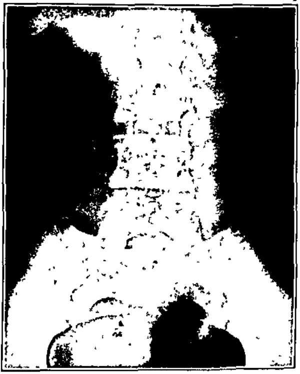


Fig. 188 —Bilateral sacro-iliac involvement with partial joint fusion Note the early ligamentous calcification at L-2 and 3 (Boland and Shebesta, courtesy of Radiology.)

third of the joint. The juxta-articular portion of the sacrum later shows

processes need not keep pace with one another, either may predominate.
At first the sacro-iliac joint appears *blurred* and the *margins* are *indis-*

ilium and sacrum appears normal or less than normal. When spotty rare-

of typhc
years.
the acti
remissions.
O
for
rela
walking until pain and muscle spasm have disappeared. In severe cases the patient is not allowed to rise for meals or to have bathroom privileges for a month or more. For cases with spinal deformity, Swaim[55] has advocated recumbency with attempts to hyperextend the spine by removing pillows from under the head and by using plaster half-shells for the back, hot fomentations for the spine and corrective exercises. After two weeks on this program, if the spine is not straightened, a light plaster jacket extending from the symphysis pubis below to the shoulder blades above is employed. The cast is not removed until all spasm has disappeared (usually several weeks); then new plaster jackets with increased correction are applied. A waxed leather jacket (laced in front and steeled in back) is then worn for prolonged periods, for several years in some cases. *But in our*

persistently carried out.

For patients with marked fixed flexion deformity of the spine surgical correction by an *osteotomy* of the laminae and articular facets has been devised. Smith-Petersen, Larson and Aufranc[51] obtained definite postural improvement in 6 cases of spondylitis with marked flexion deformity when osteotomy was performed in the lumbar region. The procedure is more favorable for the lumbar region than for the thoracic, since the latter commonly presents ankylosed costovertebral joints rendering correction difficult, if not impossible. Selection of the level at which osteotomy may be performed depends on the roentgenographic findings; the less ligamentous ossification there is, the better the chance for correction. Following surgery *a plaster shell is applied and is kept on for four to six weeks;* this is followed by a plaster jacket or a back brace which is worn *continuously* for a year or longer

OTHER TYPES OF SPONDYLITIS

The types of arthritis of the spine discussed below are not "closely allied to rheumatoid arthritis," but are included here because of possible confusion with rheumatoid spondylitis.

SYPHILITIC SPONDYLITIS

Syphilis may affect the spine directly as a syphilitic synovitis, osteitis or periostitis, or indirectly as an osteoarthropathy (*"Charcot joint"*) *secondary* to syphilitic tabes ... very rare. The reported incidence of ... philitic arthritis involving peripheral ... xact criteria for diagnosis are being us... g more *efficient* treatment.

When the disease is mild the first changes in the sacro-iliac joints usually consist of a zone of subchondral sclerosis, haziness of the joint space and loss of definition of the articular margins. As the disease progresses, iliac



FIG 189 —Involvement of the posterior intervertebral (apophyseal) joints The articular facets of L-3 and 4 show mottled rarefaction with irregularity of the joint spaces and juxta-articular sclerosis. There is already ligamentous calcification at L-2 (Boland and Present, courtesy Jour Am Med Assn)

sclerosis covers a wider zone and the juxta-articular portion of the sacrum shows similar changes The articular space gradually becomes more and more narrowed, and finally fusion occurs. Spotty juxta-articular rarefaction is rarely conspicuous and may be entirely absent

In moderately severe cases the first changes ...

joint space mottling is usually definite, and the articular margins

Syphilitic spondylitis with invasion of bone and periosteum by Tre-

vertebra or 2 or 3 adjacent ones The vertebrae may show evidence by x-ray of bone destruction or new bone production, or a combination of both Eburnation of the vertebral bodies occurs occasionally, and in most instances the vertebrae eventually develop secondary spur formations.



FIG 193 —Charcot's joints involving the third, fourth and fifth lumbar and the first sacral vertebrae

tenderness on palpation Usually there are coexisting lesions in other bones such as the sternum, clavicle, tibia or radius. Gummatous lesions of the

bone occurring simultaneously with extensive (but chaotic) new bone and cartilage production. (Fig. 193) The pathologic changes reflected roent-

are irregular. As fusion takes place bony trabeculae can be seen transversing the joint and gradually the joint space becomes irregularly narrowed. When ankylosis has developed, tell-tale evidence of spotty rarefaction may be left.

fac

an

minimal or even absent

Posterior Intervertebral (Apophyseal) Joint Changes.—The changes in the apophyseal joints are neither as constant nor as definite as those which



Fig 190

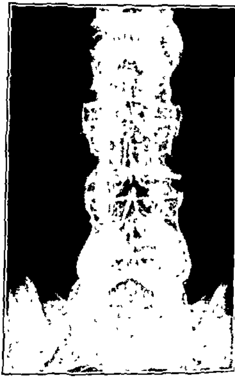


Fig. 191

Fig. 190 —Ligamentous calcification

Fig 191.—Typical "bamboo spine" and complete ankylosis of the sacro-iliac and posterior intervertebral (apophyseal) joints

occur in the sacro-iliac articulations. Even with the use of special techniques, detailed studies may yield disappointing results. Typical findings in the apophyseal joints are rarely present without accompanying evidence of sacro-iliitis. Often only a few scattered joints are abnormal while intervening ones appear normal. Presumably the pathologic process in the inter-

of the spinal cord, leprosy, etc.

As with tabetic joints elsewhere, antisyphilitic therapy is not effective Conservative orthopedic treatment with nonsurgical immobilization may be needed Surgical fusion of the involved vertebrae is not always attended by good results.

OSTEOARTHRITIC (HYPERTROPHIC) SPONDYLITIS

Osteoarthritis of the spine, the equivalent and the accompaniment of osteoarthritis of peripheral joints, represents primarily a degenerative rather than an inflammatory change. Hence, such terms as *degenerative joint disease of the spine* or *degenerative spondylosis* are more accurate and are preferred by some For the sake of uniform terminology, however, osteoarthritis of the spine or osteoarthritic spondylitis are acceptable designations.

Although exostoses at times may be of sufficient length to coalesce, ankylosis is not a feature of osteoarthritis. Degenerative processes may be found in the capsules of the small posterior intervertebral (apophyseal) joints and in the paravertebral ligaments, the latter sometimes demonstrating spotty calcific deposits.

On the basis of clinical symptoms it must be assumed that *transient reactions occur in certain soft tissues* including the paraspinal ligaments, the joint capsules and the periosteum. Such reactions would help to explain

probable that a "*secondary intramuscular fibrositis*" may also coexist, especially of the paraspinal muscles; this would explain the diffuse muscular symptoms which so commonly accompany the process.

... degenerative changes in the

reveal alterations such as marginal over-growth of bone, especially in the *lower cervical and lumbar regions. But in the majority of cases osteoarthritis of the spine does not cause symptoms When symptoms do occur* they are usually mild unless the process has been *activated* by *trauma* or is *associated* with *radiculitis*. Aching and stiffness of the neck or lower back accompanied by varying degrees of muscle spasm and restricted motion are common. The symptoms are usually mild or relatively mild and are recurrent and transient, lasting for a few days or weeks at a time. The discomfort may be rather diffuse, and with cervical involvement may extend into the occipital region of the head, the trapezius areas or shoulders. The aching and stiffness, as a rule, exhibit the qualitative characteristics of "fibrositis," being influenced by weather changes, applications of local heat, use of the part and salicylates. Forced motion of the neck is likely to aggravate the discomfort. The episodes may come on spontaneously for no apparent reason or may be provoked by trauma, often minor. With

with osteoarthritis of the lumbar spine.



FIG 194 —Osteoarthritic changes in the lower cervical spine.

The changes which accompany osteoarthritis of the spine may produce compression of spinal nerve roots and symptoms of radiculitis. Narrowing of the intervertebral foramina may result from thinning of the intervertebral disc and/or from pressure from osteophytes and their thickened overlying soft tissues (*See also* Chapter 32)

Roentgenographic Findings.—These are highly characteristic and are distinct from those of rheumatoid spondylitis. The most striking changes

being present without reduction in the size of the intervertebral spaces. Occasionally adjoining spurs may be sufficiently long to coalesce, but when

colored and white is approximately equal From 187,987 admissions to the hospital, there were 150 cases of acute rheumatic fever for an incidence of 0.08 per cent. Forty-four per cent of these were negro and 56 per cent were white The authors conclude that there is no appreciable racial difference in active and inactive rheumatic heart disease, but that the

Hedley[90] found a reduced incidence among negroes in Philadelphia

ETIOLOGY AND PATHOGENESIS OF RHEUMATIC FEVER

Factor of Infection.—(1) **Streptococcic Infection** —During the past ten years studies and reports have accumulated indicting the hemolytic streptococcus (Group A) until the evidence is quite overwhelming *It is probably universally now accepted that the Group A hemolytic streptococcus is either the direct or indirect cause, or associated with the direct onset, of rheumatic fever.*[64,74,75 103 134 135 159 180 208] During the recent war, the close relationship of epidemics of hemolytic streptococcal infections and rheumatic fever in the Armed Forces was repeatedly noted and reported[43 72,100 106 217] The carrier rate for Group A hemolytic streptococci in well troops was shown by Van Ravenswaay to parallel rather closely the incidence of rheumatic fever. Watson, Rothbard, and Swift made a careful analysis of 110 patients with definite scarlet fever, a known hemolytic streptococcal disease While only 19 patients developed definitely abnormal electrocardiograms, 8 of the 19 developed clinical attacks of rheumatic fever and the authors believe that all suffered from the same fundamental tissue injury following their streptococcal infection, namely, that characteristic of rheumatic fever[202] Rantz and associates[152] made a detailed study of the hemolytic streptococcic sore throat in a large number of individuals About 20 per cent of their patients demonstrated late complications in the way of arthritis, carditis, fever, skin lesions, prolonged elevation of the ESR, and other manifestations which they suggest grouping under the name of "Post-streptococcic state" They further emphasize the specific relationship of the hemolytic streptococcus in the etiology of rheumatic fever by again pointing out that this phase of the post-streptococcic state is one and the same identical process as rheumatic fever

parable in cases of rheumatic fever as in cases of proved hemolytic streptococcic origin[24 62 73 131 141]

(2) **Other Infections.**—No very good evidence is available that clearly ties any other type of infection closely to the onset of rheumatic fever

this occurs it is usually in an isolated region. Bambooing of the spine which characterizes an advanced stage of rheumatoid spondylitis, *does not occur.* Posterior spurs arising from the posterior margins of the vertebral bodies and/or from the margins of the articular facets are less common but of greater clinical importance. (*See also* Chapters 6 and 32.)

The degree of intervertebral joint space narrowing varies considerably.

[illegible]



Fig 195.—Osteoarthritis of the dorsal spine Note the pronounced exostoses at the anterior vertebral margins and the narrowed intervertebral spaces

Osteoporosis of the vertebral bodies does not occur except as a part of generalized skeletal demineralization incident to the aging process. Extensive calcification of the paravertebral ligaments is absent, although scattered small calcium deposits may be noted. *Definite alterations in the sacro-iliac joints are not a part of the picture of osteoarthritis;* but with advancing years there may develop some juxta-articular sclerosis and joint space narrowing together with hypertrophic spurs at the inferior borders

Other Laboratory Tests.—These are essentially normal. Evidently osteoarthritis is largely confined to the skeletal structures and signs of toxemia are absent If anemia is present it should be ascribed to other

Copeman[48] describes some instances when the infection preceding rheumatic fever appeared not to be of the streptococcic type in every case. There was some evidence that such diseases as dysentery, sand fly fever, and malaria might at times be the preceding infection to rheumatic fever At no time, however, was a hemolytic streptococcal coincidental infection thoroughly ruled out Other infections such as influenza have been clearly shown by a number of workers to have no relationship in precipitating rheumatic recurrences in children who have had previous attacks of rheumatic fever [172,156] Also Green[74] has shown the complete absence of any relationship between acute rheumatism and epidemics of rubella, measles, chickenpox, common cold, and diphtheria

(3) **Experimental Transmission of Rheumatic Fever.**—*Copeman*[47] *reported* the first probable transference of rheumatic fever from a patient with the

[illegible]

fever in 2 of them after twenty-four hours and pain in the shoulder muscles without pyrexia three days later in another. The blood from the 2 patients contracting mild rheumatic fever was pooled on the third day and again *passed on to 4 volunteers. One of the 4 developed an attack of febrile* fibrositis on the second day. The blood of the more severely affected of the first 2 cases was again transferred on the seventh day of his illness to 4 other

[illegible]

infective agent was present in the blood of the original patient which was capable of transference to others Any confirmation of this work would *offer very strong evidence toward a virus etiology.*

Factor of Vitamin Deficiency.—It was suggested that chorea may be primarily a vitamin deficiency, especially of the B-6 factor.[175] Kutumbiah[123] cited the infrequency of rheumatic fever in patients with frank avitaminosis *B* in *India as evidence against the contention that rheumatism is* related to Vitamin B deficiency. Irrespective of the concentration prior to the onset of rheumatic fever, there is a fall in the level of Vitamin A in the plasma with the development of acute rheumatic fever. The carotene content does not change *significantly.*[177] *Marked lowering of the Vitamin* C content in the plasma of rheumatic fever patients was reported.[164] He also made a preliminary report on the possible influence of Vitamin P in rheumatic fever. Peete,[148] after studying the dietary program of 50 patients *with rheumatic fever, found a deficiency in calcium, phosphorus, and* Vitamins A and D.

Physical factors such as exposure, cold, wet, and excessive fatigue are important predisposing factors, and a small series of cases in which physical *factors in extreme degree appeared to be alone responsible* was reported [47] Eleven cases were reported in which rheumatic fever began within a few [illegible] joint was the first affected [69]

[illegible] *us injections of purified* [illegible] 65 per cent of normal children and 83 per cent of rheumatic children gave a positive reaction.[186,187] Struck by the relationship of periarteritis nodosa to sensitivity states, Rich

causes. The erythrocyte sedimentation rate is within normal range. *Agglutination and precipitin tests with hemolytic streptococci are negative.* No significant changes in serum calcium, phosphorus or phosphatase are found.



FIG. 196 —Osteoarthritis (osteoarthritic spondylitis) of the lumbar spine in a seventy-three year old patient Note the marked osteoarthritic spurring with an almost complete bridge on the right between lumbar vertebrae 2 and 3.

Treatment —Treatment in general is similar to that for osteoarthritis elsewhere in the body (see Chapter 40, p. 728). Unless symptoms are present no treatment is indicated. If rather pronounced but symptomless changes are found roentgenographically it is well to caution the patient against trauma and obesity

Mild symptoms and transient recurrences of more acute symptoms usually respond to simple measures such as rest, local heat (electric pad,

and Gregory[160,161,162] produced in rabbits, with the antigen horse serum, lesions considered identical with those of that disease. By the same means

valvular nodular projections of swollen and degenerating collagen with damaged overlying endothelium; small foci of myocardial necrosis and myocardial lesions with the basic characteristics of Aschoff bodies. Such changes did not occur spontaneously in hundreds of other rabbits. *Rich*[162] *concluded that rheumatic fever is an allergic disease with a native individual*

many lesions typical of streptococcic disease of human beings and some cardiac lesions resembling those found in human rheumatic fever by injecting sterile erythrogenic filtrate of NY-5 hemolytic streptococci into young albino rabbits. Clawson failed to confirm the work of either Rich or Robinson and found no evidence favoring the theory that streptococcic toxin acts directly or by an allergic reaction on valves. Neither was he able to produce rheumatic heart lesions from hypersensitiveness or serum sickness alone. But rats infected intracardially with Streptococcus viridans or hemolytic streptococci developed acute rheumatic-like endocarditis and bacterial endocarditis or both. Clawson concluded that acute rheumatic endocarditis and bacterial endocarditis occur only as a response to a direct valvular infection with bacterial cells.

It is obvious from the above that our experimental pathologists at present are still apart in their concepts and interpretation of experimental lesions produced. *We may safely conclude that rheumatic fever has not yet been successfully produced in an animal.*

Conclusions on Etiology.—*Rheumatic fever occurs following a Group A hemolytic streptococcic infection in a susceptible individual in a climate having the necessary meteorological factors. To produce the disease, Factor X, which is at present unknown, is also probably required.*

PATHOLOGY OF RHEUMATIC FEVER

General Data.—The two basic inflammatory changes occurring in rheumatic fever are exudation and proliferation, both of which represent response of tissue to injury. The exudative process is responsible for the swollen joints and rheumatic pleurisy and the proliferative reaction for the Aschoff bodies and subcutaneous nodules.

synovial membrane.

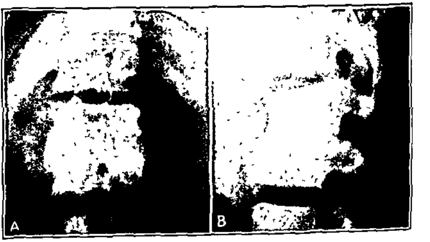
A pancarditis is usually present, involvement of the endocardium is the outstanding lesion in most cases. Myocarditis occurs in practically all

hot fomentations, infra-red radiation or diathermy), gentle massage and salicylates. For patients with chronic low back disability aggravated by weight-bearing and for those with recurrent symptoms provoked by repeated trauma (as in mechanics, industrial workers, day laborers, farmers, etc.) orthopedic supports (braces or corsets) may be required (see Chapter 19, p. 361). Corrective postural exercises and reduction in body weight are indicated for some. A firm nonsagging bed (with bed boards) and short periods of bed rest during the day are often helpful. Local infiltration of nerve roots with 1 per cent procaine solution occasionally may be helpful. Roentgen therapy provides partial or complete relief of radicular pain in a sufficient number of cases to warrant trial. With severe radicular pain, however, cervical immobilization and traction are also needed. The mechanism by which roentgen therapy produces amelioration is not known [illegible]

[illegible]

SO-CALLED INFECTIOUS SPONDYLITIS

Acute infectious lesions of the intervertebral discs, sometimes called infectious spondylitis or intervertebral discitis, have been reported following a number of diseases, including measles, scarlet fever, influenza, smallpox, typhus fever, malaria, brucellosis, typhoid or paratyphoid fever and infections with pneumococci, staphylococci or streptococci.[1] Symptoms include pain in the back, fever, spasm of the para-spinal or psoas muscles, tenderness over the affected area on percussion and palpation, and limitation of motion in the affected segments of the spine.



A B

Fig. 197*A* and *B*.—Specific infectious spondylitis involving lumbar vertebrae 1 and 2. The intervertebral disc has been destroyed and there is almost complete fusion of the adjacent vertebral bodies.

cases and pericarditis is also frequent. The endocardium covering the mitral valve is most often affected although the aortic and tricuspid valves may also be involved in the process.

The mitral valve is damaged in practically all cases of rheumatic carditis, while the aortic is injured in 50 per cent, the tricuspid in 33 per cent, and the pulmonary only rarely. Mitral stenosis and aortic insufficiency become *more frequent with an increasing number of attacks.*

Rheumatic verrucae are small, firm, wart-like excrescences most often seen at the line of closure of the valve leaflets on the left side of the heart, especially the mitral and aortic valves.[210] They average 1 to 2 mm in size and may become confluent

Active carditis occurs more frequently when polyarthritis is present at the onset and less often when the initial signs are chorea or growing or joint pains

The outstanding pathologic lesion in the myocardium is the Aschoff nodule.[3] These lesions are found most often in the posterior wall of the left ventricle and basal part of the interventricular septum and consist of large, irregularly shaped, round, wandering cells often placed concentrically

other organs

Special Clinico-Pathological Data.—Cardiovascular System.—Of 2,476 cases of rheumatic valvular disease from five large cities studied by Special

were unspecified.[130]

Hall and Anderson[81] studied 124 hearts of which 112 were free of valvular lesions as commonly understood The remaining twelve specimens were used as controls and presented gross lesions of rheumatic heart disease or sub-acute bacterial endocarditis or both. Of the 112 supposedly normal hearts, 90 per cent revealed stigmata of rheumatic infection in the myocar-

post-traumatic thinning of the disc, congenital absence of the disc, osteochondrosis of the disc and specific infectious processes. Usually it can be differentiated from tuberculosis by its more sudden and violent onset and the history of a preceding acute infectious process and rapid healing with fusion of adjacent vertebrae.

Prognosis in general is good as to recovery and usually little or no disability results. In the presence of severe symptoms with fever, marked pain and prostration, the patient is treated symptomatically and the spine is fixed by means of a plaster cast. With subsidence of pain and evidence of healing (within several months), a corset or a steel brace may be used until the symptoms have entirely cleared (within a year or so)[21]

BIBLIOGRAPHY

1 Bade, H Roentgenpraxis, **11**, 461, 1939
2 Beadle, O A Med Res Council, London, Special Rep Series No 161, 1931 The Intervertebral Discs
3 Boland, E W California Med, **65**, 285, 1946
4 Boland, E W, and Present, A J Jour Am Med Assn, **129**, 843, 1945
5 Boland, E W, and Shebesta, E M Radiology, **47**, 551, 1946
6 Boland, E W, Headley, N E, and Hench, P S Ann Rheum Dis, **7**, 196, 1948
7 Boland, E W, Stephens, C A L, and Hench, P S To be published
8 Buckley, C W Brit Med Jour, **2**, 4, 1943
9 Cecil, R L, Kammerer, W H, and De Prume, F J Ann Int Med, **16**, 811, 1942
10 Dawson, M H in Nelson's Loose Leaf Living Medicine, New York, Thomas Nelson & Sons, **5**, 605, 1935
11 Desmarin, N N Arch f klin Chir, **153**, 358, 1928
12 Dunham, C L, and Kautz, F G Am Jour Med Sci, **201**, 232, 1941
13 Fischer, A, and Vontz, O Mitt a d Grenzeb d Med u Chir, **42**, 586, 1932
14 Forestier, J Radiology, **33**, 261, 1939
15 ——— *Ibid*, **33**, 389, 1939
16 Freund, H A, Steiner, G, Leichtentritt, B, and Price, A E Science, **101**, 202, 1945
17 ——— Am Jour Path, **18**, 865 1942
18 ——— Jour Lab and Clin Med, **27**, 1256, 1942
19 [illegible]
20 [illegible]
21 [illegible] th Med Jour, **33**, 347, [illegible]
22 Golding, F C Brit Jour Surg, **23**, 484, 1936
23 Hanflig, S S Jour Am Med Assn, **106**, 523, 1936
24 Hare, H F New England Jour Med, **223**, 702, 1940
25 Hemphill, J F, and Reeves, R J Am Jour Roentgenol and Rad Therap, **54**, 282, 1945
26 Horwitz, T Arch Int Med, **65**, 1178, 1940
27 Kahlmeter, G Brit Jour Actinother, **5**, 93, 1930
28 ——— Reports on Chronic Rheumatic Diseases, London, H K Lewis and Co, Ltd, 1937
29 Kemen, A Verhandl d Deutsch Gesellsch f Inn Med Abst, **49**, 46, 1937
30 Kienbock, R Wien Arch f inn Med, **32**, 311, 1938
31 Krebs, W Fortschr a d Geb d Rontgenstrah, **50**, 537, 1934

polyarthritis preceded death by five months or less Three cases gave no history of rheumatic fever despite the presence of Aschoff bodies

Stern[181] reported Stokes-Adams attacks in a child with insidious rheumatic pericardial effusion, and Hartwell[86] reported intermittent bundle branch block in a case of acute rheumatic fever

Keefer[111] states that auricular fibrillation increases in frequency as age advances. Hutcheson[104] studied the musculature of two rheumatic hearts

concludes that auricular fibrillation in patients with chronic rheumatic heart disease may be due to substitution of the auricular smooth muscle for striated cardiac muscle.

Couper[59] noted 129 cases of pericarditis in the 1,632 cases studied Ash[7] in a study of 553 children observed for 9 6 years found pericarditis an ominous sign for 75 per cent terminated fatally

Coronary Occlusion.—Weinstein[206] emphasized the possible rheumatic
thirty years of age by
y infections, occasional
were used to establish
the suggestion of the relationship Ernstene and Schneider[60] reported 6 cases of angina pectoris in young persons with inactive rheumatic heart disease and aortic insufficiency and believed the coronary insufficiency due

"Masson body" is not specific [95] The lesions were believed to be due to capillary damage with altered permeability Disseminated nodular cal-

finding in 6 cases

prognostic sign.

Nervous System.—Breutsch[27] considered the problem of late cerebral

32. KREBS, W, and VONTZ, O.: Deutsch med. Wchnschr., 60, 100, 1934
33. KUHNS, J G New England Jour Med, 217, 317, 1937.
34 LERICHE, R, and JUNG, A: Lyon chir, 28, 408, 1931.
35 LUDWIG, A O., SHORT, C L, and BAUER, W.. New England Jour. Med, 228, 306, 1943.
36 MARIE, P Rev. de med, 18, 285, 1898
37. MCFARLAND, J. W., and KRUSEN, F H: Arch. Phys Ther., 24, 263, 1943
38 MILLER, J L Jour Lab. and Clin Med., 22, 10, 1936.
39 NACHLAS, I W Jour Am Med Assn, 103, 323, 1934.
40 OPPENHEIMER, A Am Jour. Roentgenol and Rad. Therp, 49, 49, 1943
41 POLLEY, H F *Thesis, Graduate School, Univ. of Minn, 1945.*
42 SAVAGE, O: Brit Med. Jour, 2, 336, 1942
43. SCHMORL, G, and JUNGHANNS, H Die gesunde und kranke wirbelsaule im Rontgenbild, Leipzig, G Thieme, 1932
44 SCOTT, S G Acta Rheumatol, 6, 7, 1934
45 ——— *in* Charterhouse Rheumatism Clinic Original Papers, London, Oxford Medical Publications, vol 1, 1937
46. ——— Wide-Field X-Ray Treatment, London, George Newnes, Ltd, 1939
47. ———: A Monograph on Adolescent Spondylitis or Ankylosing Spondylitis The Early Management and Its Treatment by Wide-Field X-Ray Irradiation, Oxford Univ. Press, London, 1942
48 SHORE, L R Brit Jour Surg, 22, 850, 1935
49 SMITH, J R., and KOUNTZ, W B. Ann. Int Med, 17, 604, 1942
50 SMITH, R T., BOLAND, E. W, SHEBESTA, E M., and HENCH, P. S: In press
51 SMITH-PETERSEN, M. N, AUFRANC, O E, and LARSON, C. B Jour Bone and Joint Surg, 27, 1, 1945
52 SMYTH, C J, FREYBERG, R H., and LAMPE, I: Jour. Am. Med. Assn, 117, 826, 1941
53. SMYTH, C J, FREYBERG, R H, and PECK, W S: Jour Am Med Assn, 115, 2209, 1940
54 SOL, A Rev franc de dermat et de venereol, 8, 581, 1932.
55 SWAIM, L T Jour Bone and Joint Surg, 21, 983, 1939
56 SWAIM, L T, and KUHNS, J G. Arch Phys Ther, 13, 517, 1932
57 TYSON, T L M. Clin North America, 21, 1755, 1937.
58 WOLFF, C: Ztschr f orthop Chir, 63, 133, 1935
59 BOLAND, E W., and HEADLEY, N. E: J. A. M. A., 144, 365, 1950
60 ———: J A M A, 145, 8, 1951
61 BOLAND, E W Brit Med J., 2, 191, 1951
62 HERSH, A H, STECHER, R M, SOLOMON, W M, WOLPAW, R, and HAUSER, H. Amer Jour Human Genetics, 2, 391, 1950
63 ROGOFF, B, and FREYBERG, R. H Ann Rheumat. Dis., 7, 40, 1948

only 1.7 per cent in dementia [illegible]
unsuspected rheumatic heart [illegible]
not unlike that noted in the s[illegible]
as rheumatic encephalitis and cerebral embolism. The term "rheumatic epilepsy" was suggested for those cases who exhibit convulsive seizures following rheumatic fever or chorea and for those epileptics who have chronic rheumatic valvular disease.[28] Foster[65] *noted 29 cases of rheumatic* heart disease associated with convulsive disorders without other known etiology. Of these 58 6 per cent had had Sydenham's chorea previously. Dublin[55] noted that in some psychotic persons having rheumatic endocarditis often recognized only at autopsy focal and diffuse cerebral vascular lesions occur High and Aegerter[95] noted a case of rheumatic fever proven at autopsy in which meningoencephalitis was found. Changes compatible with the inflammation of rheumatic fever were found in sections of the brain and meninges. Warren and Chornyak[199] reported 5 cases of rheumatic encephalitis with case histories.

LABORATORY FINDINGS

1. Rapid sedimentation rate
2. Leukocytosis with shift to the left in the differential white blood count

[illegible]

5. Blood cultures usually negative
6. Anemia (secondary) in severe cases.

In a comparison of several tests upon patients with rheumatic fever, the *corrected sedimentation rate was found to be the most sensitive, the formol-*gel test the least sensitive, and the Weltmann reaction intermediate in sensitivity.[120]

An increase in the rapidity of the sedimentation rate is the best index of persisting infection. In general, the patient should be kept in bed until the sedimentation rate has returned to normal or nearly normal.

Antifibrinolysins.—Hemolytic streptococci will liquefy normal human fibrin clot, but the plasma clots from the blood of patients convalescent from acute hemolytic streptococcal infections resist this fibrinolytic action,[136] the reaction, however, is not specific.[193] The presence of antifibrinolysins indicates a recent hemolytic streptococcal infection and is shown by the fact that the fibrin in blood serum shows resistance to lysis when exposed to cultures of hemolytic streptococci, *i e*, its clot is not liquefied by these organisms. It is of academic interest only that such antifibrinolysins are present in the blood stream of patients with rheumatic fever, but are absent in those with rheumatoid arthritis.[80,143]

Boisvert found the fibrolysin test positive 68 times in 76 children with rheumatic fever.[24] The test usually becomes negative with the onset of convalescence. The fibrolysin test may also be positive in streptococcic cervical adenitis and in hemorrhagic nephritis The behavior of this reaction is added evidence of the etiologic significance of hemolytic streptococci *in rheumatic fever.*

Antistreptolysins.—Synonyms for this include antihemolysin and antistreptohemolysin. A streptolysin, which is a soluble product of hemolytic

CHAPTER 32

RHEUMATIC FEVER

By W PAUL HOLBROOK, M.D

IMPORTANCE

ause of disability and death throughout later life Rheumatic fever is the ommonest cause of organic heart disease under the age of forty. World Var II demonstrated that rheumatic fever is not only a serious childhood isease but it can attack adults in epidemic proportions. More than 40,000 ervicemen contracted rheumatic fever during the War and lost in excess of ,000,000 man days of service The cost to the Government for time lost, ospitalization, convalescent care, and pension disabilities is a staggering um

INCIDENCE

Exact determination of the incidence of rheumatic fever continues to be andicapped by the fact that the disease is not reportable in most parts of he world However, a number of useful and reasonably accurate estimaions are available It is estimated by Keith[115] that rheumatic heart disease ffects 0 10 to 2 08 per cent of the school population in Great Britain, 0 9 to 36 per cent in the United States, and 0 36 to 3 92 per cent in Canada In New York City from 1936 through 1938, there were 681 deaths from rheunatic fever—or rheumatic heart disease—recorded in children between the ges of five and fourteen years [133] This exceeded the deaths from accidents or that age group and was two and one-third times greater than deaths from nfantile paralysis pulmonary tuberculosis, scarlet fever, measles, whoopng cough, and diphtheria combined Martin also estimated that there were one million people in the United States with rheumatic heart disease,

streptococci, produces hemolysis of red blood cells It is an antigen, and as such leads to the formation of an antibody known as antistreptohemolysin or antistreptolysin, which latter may neutralize the streptolysin. The titer of antistreptolysin in a given serum may be determined by titrating with streptolysin. Antistreptolysins are present only following hemolytic streptococcal infections. Patients with active rheumatic fever usually show a high antistreptolysin titer Again, this is of academic interest only, as the test is not simple enough to be utilized generally

Antihyaluronidase Titer.—Recent observations[222-226] on streptococcal antihyaluronidase in human sera have shown that the concentration of that antibody is elevated above the normal in acute rheumatic fever, after streptococcal infection, in acute glomerulonephritis, and even in rheumatoid arthritis. The degree of elevation is much greater, however, in acute rheumatic fever and in glomerulonephritis than in the other conditions The titer may be determined either by the mucin-clot prevention method,[223] or by the turbidometric method of Harris.[225] By either technic the serum from a patient with rheumatic fever shows a titer of 1:128-1:512, with the mean at 1:360 In rheumatoid arthritis the titer is less than 1:128, with the mean at 1:74 The test has proved an accurate index of occurrence of rheumatic fever in large series of cases, and is now accepted as a standard diagnostic criterion generally [226]

Precipitins.—In acute rheumatic fever, the patient develops precipitins to protein fractions of hemolytic streptococci Hemolytic streptococci have

(M and S)

Blood Phosphorus.—The phosphorus content of whole blood of children with rheumatic fever was found to be about 15 per cent less than patients convalescing from other acute and chronic illnesses There is normally a high requirement of phosphorus for optimal growth Phosphorus also plays a rôle in muscle metabolism [69]

Cholesterol.—Offenkrantz and Poindexter noted hypocholesterolemia in acute rheumatic fever and rheumatic carditis in some cases [144 150]

Capillary Resistance—Brown and Wasson tested 150 children with

Electrocardiogram.—Reyersbach and Kuttner concluded that prolongation of the P-R interval was not in itself a reliable index of myocardial involvement inasmuch as they found an abnormal prolongation of the auriculoventricular conduction time in 5 per cent of 150 normal girls whose electrocardiograms were studied [155] Abnormalities were noted in 50 to 77 per cent of cases having acute rheumatic fever in 6 series reported

Heffer noted abnormalities in 39 per cent of electrocardiograms made on 181 patients in whom Sydenham's chorea was the first sign of rheumatic fever [92] These changes occurred in the absence of clinical or other laboratory findings of cardiac involvement Atropine cannot be used to differen-

500,000 children handicapped by rheumatic fever and rheumatic heart disease in this country.[128]

There has been a decline in England from 23 deaths per million in 1939 to 12.1 in 1942 [70] The Metropolitan Life Insurance Company[138,139] reports a decrease in deaths from rheumatic fever and rheumatic heart disease between the ages of five and twenty-four from 32 3 per 100,000 in 1917 and 1918 to 9.7 in 1943 Ash[5] states that each five-year period since 1922 has shown a lowered mortality rate and a decrease in the percentage and severity of cardiac damage

PREDISPOSING FACTORS GOVERNING INCIDENCE

Geography and Climate.—There is general agreement that rheumatic fever is more common and severe in temperate than in warmer climates The influence of geography and climate may be investigated by several

quency

southern

posts sc

incidence of the disease in the Rocky Mountain area, Great Lakes area, and mid-west, with an extremely low incidence along the Mexican border and Gulf of Mexico [99,100]

Climatic factors are exceedingly complex and comprise many other factors than latitude This is illustrated by a careful study of the incidence of rheumatic fever in school children in three California communities with different climatic factors Eureka, a northern California sea coast town with a fairly high rainfall but equitable temperature, showed an incidence of 2 per cent rheumatic heart disease in school children. Susanville, at an altitude of 4,268 with a moderate rainfall, showed an incidence of 1 1 per cent rheumatic heart disease, while Redlands, with an altitude of 1,350 feet and relatively dry, showed an incidence of only 0 38 per cent rheumatic heart disease.[174] Paul[147] found 10 times as much rheumatic heart disease among the Indian school children near the Canadian border as was present near the Mexican border.

The data on comparison of hospital admissions is summarized here. The percentages of children with rheumatic fever or rheumatic heart disease of total children admitted were as follows.—

	Per cent
Philadelphia, Pa	5 8
Portland, Oregon	3 5
Cincinnati, Ohio	2 7[203]
Dallas, Texas	0 8[63]
Los Angeles, Calif.	0.69[71]

tiate between the prolongation of the P-R interval caused by vagotonia and rheumatic fever because atropine reduces the P-R time equally in both.[176]

RELATIONSHIP OF RHEUMATIC FEVER TO OTHER DISEASES

Rheumatoid Arthritis.—The relationship of rheumatic fever to rheumatoid arthritis has been the subject of conflicting reports. At necropsy evidence of rheumatic heart disease was found in 16 (53 per cent) of 30 rheumatoid patients, according to Baggenstoss, Rosenberg and Hench[8,9,m] who concluded. "This evidence suggests ever more strongly that rheumatoid arthritis and rheumatic fever are in some manner closely related." A similar conclusion was made by Young and Schwedel[218] who found "rheumatic heart disease" in 25 (66 per cent) of 38 necropsies in cases of rheumatoid arthritis But Bennett[18] arrived at quite a different conclusion from his study of necropsies on 101 patients with rheumatic fever and 48 patients with rheumatoid arthritis: "It is apparent that the usual anatomical changes observed in rheumatoid arthritis differ so markedly from those of rheumatic fever that one must infer that the pathogenesis of the observed lesions is different."

Subacute Bacterial Endocarditis.—The recurring arthralgia and arthritis seen in some patients with endocarditis caused by *Streptococcus viridans* were considered as being due to complicating acute rheumatic fever or the "general toxemia caused by bacterial endocarditis." The latter was held more likely in view of the unusual clinical features and the failure to respond to the usual forms of therapy for rheumatic fever.[27]

In 25 per cent of 452 fatal cases of rheumatic heart disease, postmortem evidence of bacterial endocarditis was noted,[67] and rheumatic carditis was present in 90 per cent of 174 consecutive adult patients with subacute bacterial endocarditis.[88] The rheumatic heart lesion was called the "determinative background for bacterial endocarditis "

Erythema Nodosum.—No evidence supported the theory that erythema nodosum is a manifestation of acute rheumatism. Perry[149] found only 10 cases of erythema nodosum in more than 1,000 patients with acute rheumatism.

Chorea.—Sydenham's chorea is a nervous manifestation whose morbid anatomy is very imperfectly understood and whose exact relationship to other rheumatic conditions is still disputed by some.[91] Osler found acute endocarditis with great frequency in chorea and stated that there was no known disease in which endocarditis was found so constantly postmortem as in chorea.[115] Various authors have reported from 3 to 77 per cent of cases of Sydenham's chorea to manifest signs of rheumatic heart disease Hedley has summarized these.[91] In recent years, some writers have questioned whether chorea without clinical or laboratory evidence of infection should be regarded as part of the rheumatic syndrome, or at least more than a rather mild rheumatic manifestation accompanied very infrequently by cardiac damage. Hedley concluded that heart disease was less likely to develop in Sydenham's chorea unattended by other signs of rheumatoid infection. He found that 20 per cent of children with rheumatic heart disease have histories of chorea

In a large series of cases studied by Hedley, approximately 90 per cent of the admissions for chorea were confined to the five to fourteen year age

Decherd and Herrmann at Galveston, Texas[52] report that rheumatic heart disease during 1930 to 1939 constituted only 2.1 per cent of total cardiac admissions. They contrast these low findings with the figures of Chávez[34] in Mexico who reports that in charity cases rheumatic heart disease constituted 62 per cent, and in private cases 41 per cent of the total heart cases. "These are tremendous differences for which some explanation must be sought."

Comparable necropsy studies reported from various geographical areas are summarized giving the number of necropsies and the number having rheumatic heart disease with the percentages:—

	Per cent
Boston	8,300—456 or 5.5[67]
Atlanta	1,754— 61 or 3.5[39]
Galveston	2,463— 22 or 0.9[52]
New Orleans	16,121—102 or 0.63[31]
Panama	8,905— 8 or 0.089[85]

Four times as much mitral stenosis was found in Atlanta as was found in New Orleans even though the economic level of the patients in the two hospitals studied was comparable.[39] A marked increase in the incidence of rheumatic fever and rheumatic heart disease was reported in Panama even though of 228,748 hospital admissions rheumatic fever occurred in only 80 patients and rheumatic heart disease in 56, for a total of 136 or 0.052 per cent—an incidence about one-fourth that of Galveston which has an extremely low incidence.

Recent reports from Panama, South Africa, South Pacific, Ceylon, India, and various sub-tropical areas indicate the world-wide distribution of the disease. It is probable that no climatic area is entirely free. *It does seem clear, however, that the incidence varies tremendously with climatic and meteorological factors.*

Season.—In the temperate zone the greater prevalence of rheumatic fever occurs in winter and spring. No seasonal variation could be made out in India or in Panama.

Social and Hygienic factors.—The greater frequency of rheumatic fever in low-income and poverty-stricken groups was reported by a number of observers. It is possible, however, that the increase is due to crowding which was clearly demonstrated during World War II.

Family, Heredity, and Constitution.—The old argument as to the relative importance of heredity versus environment in the development of rheumatic fever continues to be a source of sharp disagreement. The familial tendency of rheumatic fever has been strongly supported for many years by Wilson[212] and Wilson, Schweitzer and Lubschez[211] in pointing out that the distribution of cases of rheumatic fever studied by them has followed the [illegible]

agreed that chorea is infrequent under the age of five

A child with almost any form of Sydenham's chorea stands a much greater chance of developing rheumatic heart disease than a child who has never had any form of Sydenham's chorea From 10 to 15 per cent of cases of rheumatic heart disease give histories of chorea, many with frank attacks of rheumatic fever.

Rheumatic fever, Sydenham's chorea, and rheumatic heart disease are

year age period

In Great Britain, most writers have indicated that rheumatic fever and chorea occurred with greatest frequency in the fall and during December

Wilson found that chorea was the initial manifestation of the rheumatic process in about 25 per cent of 699 rheumatic children. The earliest age of onset of chorea was twenty months and the oldest patient with recurrences of chorea was eighteen years Recurrences of chorea over the age of twenty are very uncommon

Prodromal symptoms of chorea include personality changes, frequent emotional upsets, enuresis, restless sleep, etc A very obedient child may become irritable, inattentive, clumsy and sulky, or may act silly These individuals may then show the incoordinate, irregular, objectively purposeless movements which may become severe enough to interfere with walking, talking, and swallowing They are usually relieved by sleep, rest, hot

turbances

Chorea has been noted as a symptom of rheumatic fever in from 5 to 34 per cent of cases [209] The commonest cause of chorea in childhood is a rheumatic infection Chorea is nearly twice as common in females as in males [89] Sydenham's chorea is relatively uncommon among negroes. Children with rheumatic fever are no more nervous than those with other diseases Some workers[43] feel that chorea and rheumatic fever are independent while others maintain that chorea is merely a symptom of rheumatic fever

In uncomplicated cases of chorea (*i e.*, without other evidence of rheumatic activity) the sedimentation rate has been reported by some to be normal,[82] while Bauer found a rapid rate in 100 per cent of 70 choreic patients upon admission [84]

Jones and Bland found that only 3 per cent of 134 patients experiencing chorea alone had heart disease, but Wilson found no significant difference

been unable to find any significant familial incidence by the methods used by them. Guy[79] in 178 patients with rheumatic heart disease found others in the families involved only 5 times. Davis and Rosin[51] in 157 cases in Los Angeles found 8.9 per cent familial incidence. Among 211 children with rheumatic heart disease, only 5.7 per cent with a positive family history were found, whereas 3 6 per cent of the normal children gave a positive family history.[174] Kutumbiah[123] *in India among 457 cases of rheumatic* heart disease found but seven with a positive family history. Weiss[207] in Louisville, Kentucky, among 151 patients with rheumatic heart disease *encountered no other cases in siblings.* Keith and Ross[117] in Canada among 103 cases of rheumatic heart disease reported a 12 per cent positive family history. Copeman[48] in England reported that of 115 children with acute rheumatic fever 7 6 per cent of their parents gave a history of having rheumatic fever. An important and recent contribution to the problem of heredity versus environment has been made by Griffith, Moore, McGinn, and Cosby.[77] After a careful study of a group of 3,594 young adults with

not after he is separated from the family There does not, therefore, appear to be a strong and inherited susceptibility. The occurrence of multiple cases in families could be explained either on the basis of common environment or contagion. In the light of the widely accepted relationship of streptococcal infection to rheumatic fever, and the lack of agreement concerning the rôles of social, economic, and geographic factors, we prefer to look on these data as indicating the dominant rôle of contagion in the *development of rheumatic fever* "

Paul[146] and Massell and Jones[134] state that the familial factor in rheumatic fever is similar to that in tuberculosis.

One may fairly conclude that the evidence for environment and contagion as contributing factors in rheumatic fever is overwhelming, while the evidence for susceptibility, being wholly an hereditary factor, is not as yet acceptable Furthermore, the problem of environment and contagion is vulnerable to attack in the prevention of the disease and offers a positive approach, whereas very little can be done about hereditary factors in a constructive way *For this reason the emphasis should now certainly be placed upon the*

s, if one excludes of 3,129 patients imately the same e Company[138,140]

reports the death rate in males to be one-fifth greater than in females Lake[121] gives the ratio of rheumatic heart disease as greater in males in the ratio of four to three

Age.—Cohn and Lingg[44] found the peak incidence of onset at the age of eight Jones and Bland[107] in an analysis of 1000 cases stated that the average age of onset was eight. Rakov and Taylor[151] reported the oldest acute rheumatic fever in a patient of sixty-one years of age, and the youngest case was reported in a new-born infant ten days old.[54]

Race.—Bruno and Engelhardt[31] reported a comparative study from Charity Hospital in New Orleans where the admittance ratio between

be removed from school and placed at rest in bed for several weeks with mild sedatives and salicylates. Chorea appears to be a self-limited disease and is rarely fatal.[210]

Criteria of effective therapy are (1) whether the duration of the disease has been shortened and (2) whether rheumatic heart disease has been prevented. Artificial fever therapy was considered almost specific. In 17 cases ty

tures of

E. L. B

sults.

increase the hazard of treatment. Chorea rarely recurred after a full course

22 per cent of cases of rheumatic endocarditis by one individual,[15] while others stress its infrequency (1.3 per cent) Uremia has occurred in rheumatic fever, with recovery following appropriate therapy.[176]

There have been numerous observations of the infrequency of clinical evidence of renal involvement during the active stage of rheumatic fever; this varied from 0.67 to 7 per cent. Bell found rheumatic disease and nephritis frequently in autopsy material (with renal involvement in 25 per cent of 104 cases at necropsy which showed rheumatic valvulitis with active lesions).[15] In one institution, although 1,622 patients entered the hospital with the admitting diagnosis of "acute rheumatic heart disease," the clinical diagnosis of rheumatic nephritis was not made.[105] Three thousand necropsies were performed at this institution, among which 153 presented rheumatic cardiac disease as the principal cause of death. In 2 cases, renal involvement was probably the most significant cause of death.

Numerous observations have been made at autopsy of the frequency of glomerular and vascular changes in patients who have had rheumatic fever. Klotz emphasized the frequency of visceral involvement.[121] He described

some cases of malignant nephrosclerosis[61] Others also described renal lesions in patients with rheumatic heart disease, with the nephritis preceding, occurring simultaneously with, or following the onset of rheumatic manifestations. The glomerular lesions were usually of the focal embolic type or acute diffuse glomerulonephritis. Longcope has denied the specificity of the renal lesions and stated that they may be caused by inflammatory factors of diverse etiology.[137] It is possible that they may be the result of anaphylactic hypersensitivity.[160]

Infantile Leukemia Resembling Rheumatic Fever.—Joint symptoms

children, and the tendency of children to respond to an infection with a lymphatic reaction

Joint pains have been noted so frequently in infantile leukemia as to

ing administration of salicylates.

Smith has explained the bone and joint pains in infantile leukemia on an anatomic basis due to the invasion and multiplication of leukoblastic cells within the bone marrow During the first several years of life, actively functioning red marrow fills all the long bones The conversion of the active red marrow to nonfunctioning fatty marrow begins at about seven years of age and is complete at about twenty-one years (except for areas of red marrow in the upper ends of the proximal long bones) The bones become rarefied and leukemic cells spread beyond the bony cortex and may elevate

these diseases are indistinguishable from the manifestations of rheumatic fever In a series of 13 patients with a proven diagnosis by biopsy or autopsy of disseminated lupus or polyarteritis (periarteritis nodosa) seen by Holbrook and Stephens,[101] 11 had received previous treatment for rheumatic fever or rheumatoid arthritis As has been pointed out previously, Rich believes that the basic lesion of polyarteritis and rheumatic fever are perhaps identical

DIFFERENTIAL DIAGNOSIS

Data to be considered in the diagnosis of rheumatic fever were tabulated by Jones[108] who listed 5 major manifestations (carditis, arthralgia, chorea, subcutaneous nodules, and febrile rheumatic recurrences) and 7 minor manifestations (fever, abdominal pain, precordial pain, rashes, epistaxis,

27

order of frequency were· abdominal pain with possible appendicitis, acute anterior poliomyelitis, acute osteomyelitis, skin eruptions, and nephritis.

Perhaps the most common confusion in young adults is in the differential

tainty.

The Abdominal Pains of Rheumatic Fever.[2, 19, 134]—There are no abdominal signs which warrant clear-cut differentiation between suppurative appendicitis and rheumatic "pseudo-appendicitis," but estimations of the

years could find only two factors which influenced the risk of future recurrences and they were the age of the child and the interval of time elapsing since the last attack. It was calculated that the risk of recurrence during the year immediately following a major episode is 38.7 per cent compared to 11.2 per cent in the year following one year of freedom. Similar figures were presented by Ehlertsen,[57] *but Cohn and Lingg*[44] *in their analysis of* over 3,000 patients concluded that recurrences are most prevalent before puberty regardless of the time interval. A serious prognosis is suggested by the occurrence of. carditis, subcutaneous nodules, pneumonitis, enlargement of the heart, especially if progressive, bradycardia, and progressive and recurrent rheumatic infection

In individuals who had rheumatic manifestations but no heart disease, rheumatic heart disease developed subsequently in 25 per cent of children and in 5 per cent of adults. Seventy-five per cent of children had one or more recurrences of rheumatic infection within five years of its onset when sulfanilamide prophylaxis was not used In 70 per cent of adults with rheumatic heart disease, the lesion was slowly progressive whether it was obviously or obscurely active.

In children with rheumatic heart disease, 4 per cent died in the first attack, 12 per cent in the first five years, and from 20 to 30 per cent within *ten years of the onset of heart disease In the adult clinic patients, 40 per* cent died in ten years, and 75 per cent in twenty-three years In about 10 per cent, clinical signs of rheumatic valvular disease disappeared over the subsequent ten years.

Factors which influenced the prognosis were progressive disease, increase in the size of the heart, progressive valvular damage, pericarditis, valvular lesions, and nodules

Important Signs of Activity of Rheumatic Fever in Children	
Pallor.	Fever
Failure to gain weight	Symptoms sore throat, joint pains, etc
Rapid sedimentation rate	Cardiac murmurs
Leucocytosis.	E C G changes

Almost 50 per cent of all patients (and 85 per cent of children) with rheumatic fever have recurrences within a period of several years. Evidences of activity of the disease which should be watched for in patients who have had rheumatic fever include long-continued fever, muscle or joint pains, epistaxis, subcutaneous nodules, failure to gain weight or persistent underweight, pallor, increased sedimentation rate, leukocytosis, lassitude, tachycardia, abdominal pain, nausea, and electrocardiographic findings. [illegible] present [illegible] rheu- [illegible] until proved otherwise.

Active carditis appears to be the only manifestation of rheumatic activity of prognostic significance.[210] Ungerleider has discussed the relation

	Rheumatic Fever vs.	Rheumatoid Arthritis[14]
Age incidence	Practically 90 per cent of cases begin under 15 yrs.	Practically 90 per cent of cases begin over 15 yrs
Joints involved	Migratory	Progressive
Permanent joint damage	Uncommon, almost never	Very frequent
Response to salicylates	Usually dramatic	Slight, only temporary relief
Electrocardiogram	May occasionally show evidence of cardiac involvement	Usually negative (unless patient is of middle age or above)
Involvement of pleura or lungs	Not uncommon	None
Roentgen-rays	Usually negative except soft tissue swelling	Characteristic; may be negative early, later generalized osteoporosis, etc
Agglutination reaction with hemolytic streptococci	Usually negative	Strongly positive in most cases after several months
Antistreptolysin titer	Increased	Normal
Antihyaluronidase titer	Markedly increased >1 256	Increased <1 128

Clinical Picture of Rheumatic Peritonitis[216]

A young patient, with *previous rheumatic history* (as a rule)
Signs of rheumatic valvular damage often found in cardiac examination
Abdominal pain and signs of peritoneal irritation appearing during or just prior to the onset of an attack of rheumatic fever
Severe general reaction, *high fever and leucocytosis* (often above 20,000)
Diffuse abdominal phenomena with less tendency for localization in the right lower quadrant than in appendicitis.
Most common findings at operation or necropsy clear fluid in the abdomen, hyperemia, edema and infiltration of the peritoneal and subperitoneal tissues without evident intra-abdominal cause.

SYMPTOMS OF RHEUMATIC FEVER

No one clinical sign or laboratory test is pathognomonic of this disease.[93] The onset of an attack of rheumatic fever may be insidious or abrupt It frequently follows a sore throat or an acute tonsillitis or an upper respiratory infection. Within a short time (averaging three weeks, but varying from a

of heart size to prognosis.[101] In 246 children with active carditis, 36 per cent died and 50 per cent showed mitral stenosis with or without aortic insufficiency with at least moderately enlarged hearts; active carditis *included any active rheumatic process in the heart, clinically recognizable.* In another group of 427 children with rheumatic fever, active carditis was never recognized; only 2 of these died (1 of subacute bacterial endocarditis and 1 during childbirth). Forty-three per cent of this group developed moderate cardiac enlargement and 30 per cent mitral stenosis. Whether polyarthritis, chorea, or growing and joint pains characterized the course caused no significant difference in the degree of cardiac involvement.

Boyer and Nadas have discussed the ultimate effect of pregnancy on rheumatic heart disease.[25] Hamilton and Thomson have surveyed the *immediate risk of pregnancy* in women with rheumatic heart disease.[87] It is agreed that the woman with rheumatic heart disease who has decompensated faces a definite immediate risk in pregnancy. However, if the mother survived single or multiple pregnancies, Boyer and Nadas concluded from a statistical study that the pregnancy had no delayed deleterious effect on the course of the rheumatic heart disease. Postmortem data did not reveal that the increased load of pregnancies and motherhood produced any appreciable increase in cardiac hypertrophy There was no evidence to show that multiple pregnancies tended to lower the cardiac *efficiency permanently.*

Hoffman and Jeffers studied 61 fatalities in rheumatic heart disease complicating pregnancy and found that the fatalities resulted principally from congestive heart failure following delivery at or near term.[99] The most important factor amenable to control was the cardiac status of the patient at the time of delivery.

In one large series of rheumatic children, congestive failure occurred in 26 per cent; the mean duration of life following the attack of cardiac failure [illegible] *rheumatic child has*

[illegible] italization materially decreased the death rate nor did it increase the number of patients who were able to lead active lives in later years, this conclusion was reached after reviewing fifteen years' experience at the Children's Heart Hospital of Philadelphia.[182]

In summary, the outlook in the past for rheumatic fever victims has been tabulated by Levine[129] as follows.

> *Approximately one-third of the patients with rheumatic heart disease* [illegible] ardiac damage.
> [illegible] years of life.
> [illegible] enty-five and fifty.
> [illegible] atic carditis is thirty
> *years*

This is indeed a rather grim outlook. However, it is very probable that with full utilization of our present imperfect knowledge of the disease, particularly in prophylactic and preventive methods, recurrences will be reduced and the over-all picture of the disease materially brightened.

few days to five or six weeks) following the latter, the patient develops pain or stiffness in one joint or another, a moderate degree of fever, excessive perspiration, and tachycardia out of proportion to the degree of pyrexia.

In the average patient with rheumatic fever, an acute infection of the upper respiratory tract is followed by a silent period without symptoms for about seven to twenty-one days after which the acute febrile migratory polyarthritis appears. This is initiated only occasionally by a chill. The joints may be red and swollen, and pain and tenderness are usually out of proportion to the objective findings. Subcutaneous nodules around the joints appear in from 5 to 10 per cent of cases.

Common Symptoms of Rheumatic Fever
Frequently overlooked prodromes. marked pallor out of proportion to the anemia, ready fatigue, joint and *muscle pains*, "*growing pains*," failure
[illegible]
may be minimal or absent
Fever—101° to 104° F (oral), irregular, often between 99° and 101° F
Excessive perspiration during acute attacks
Tachycardia out of proportion to the degree of fever
Subcutaneous nodules (occasionally)
Cardiac involvement, to some extent, in practically every case
Migratory, pulmonary hemorrhagic solidification and hematuria

Loss of weight may be marked. Transient pulmonary symptoms may [illegible]

clinic. Most attacks of rheumatic fever begin in the early spring, the season of most frequent respiratory infections. In more than half of the cases, the attack is preceded by tonsillitis, sinusitis, or an acute sore throat. *Rheumatic fever is a disease mainly (but not solely) of temperate climates and is* associated in some way with hemolytic streptococcal infection.

The larger joints are most often affected first, and those of the lower extremity somewhat more frequently than the upper. The knees, ankles, shoulders, and wrists are most often involved, but the hips, elbows, and small joints of the hands and feet may be affected. No joint has seemed to [illegible]

symmetrical large joints are involved simultaneously, and the same joint may be involved several times before the infection has been overcome.

The affected joint is hot, swollen, red, and tender, motion produces marked pain. Joint pains are often more marked at night. The joint involvement may occasionally be so persistent and marked that it simulates acute rheumatoid arthritis. After a period of from two to ten days of treatment, the inflammatory changes disappear from an involved joint

TREATMENT OF RHEUMATIC FEVER

The advent of ACTH (corticotropin) and cortisone in 1949 created a tremendously increased interest in the treatment of acute rheumatic fever and also brought with it an equally great amount of confusion. Despite early high hopes it is now definitely known that these substances do not cure rheumatic fever. They appear to act by temporarily suppressing inflammatory reaction without in any way eradicating the underlying disease potential The exact mechanism by which they act is as yet unknown

It should be clear as a general principle that if a self limited acute process could be suppressed for the expected duration of illness a favorable result might be obtained. On the contrary it is well known that when such a suppression occurs by means of ACTH or cortisone in rheumatoid arthritis and other chronic diseases the disease usually recurs promptly on discontinuing the medication Rheumatic fever behaves in a similar way if medication is withdrawn before the attack has spontaneously subsided Because of the difficulties, hazards and doubtful effectiveness of long term administration of ACTH and cortisone their usefulness in rheumatic fever is limited to early treatment of the acute monocyclic attack which is self limited in duration, and to severe carditis

Indications for ACTH or Cortisone in Rheumatic Fever

1. **Severe Carditis.**—Rheumatic fever as such does not carry a very high mortality rate. Figures released by the National Heart Institute in 1951 indicated that the rheumatic fever deaths were less than one-twentieth of those caused by chronic rheumatic heart disease The joint manifestations and discomforts of the patient are usually well managed on salicylate routine Why then should ACTH or cortisone be considered? Reduction in the small number of deaths from carditis in acute rheumatic fever is certainly important, but the major problem is the prevention or reduction of the large number of individuals developing chronic rheumatic heart

manifested by tachycardia, pericarditis, gallop rhythm and congestive failure due to acute myocarditis is altered favorably and usually dramatically by cortisone or ACTH. Severe carditis then represents a definite indication for ACTH or cortisone therapy

2. **Prevention of Chronic Rheumatic Heart Disease Following Rheumatic Fever.**—A method of treatment which would prevent the development of chronic rheumatic heart disease as a sequel to rheumatic fever would undoubtedly be the greatest advance in rheumatic fever therapy since the introduction of the salicylates Past experience however indicates that rheumatic heart disease may not become clinically apparent for several years after the attack of rheumatic fever To properly evaluate results will require careful investigations of large numbers of cases for a period of five or ten years. Obviously the final answer is not yet available

The duration of the disease and degree of carditis before beginning

and the appearance of that [illegible]

[illegible]

ranging from 100° to 104° F. [illegible]
variable period of time; whe[illegible]
usually falls slowly by lysis [illegible]
Together with the fever there occurs copious sweating. This may drench the patient so that the bed clothes become soaked with perspiration Rheumatic fever is one of the so-called sweating diseases along with undulant fever, malaria, and septicemia. The pulse is markedly elevated and remains high even after the temperature falls to normal This tachycardia may persist to some extent for weeks or months.

Rheumatic fever may simulate rheumatoid arthritis so closely that only the passage of time and subsequent developments permit a correct diagnosis in some cases. Rarely, it may mimic polyarticular gout or early acute specific arthritis

Symptoms in Children.—In children, the onset of the disease is often much more insidious than in adults, and is frequently associated with upper respiratory tract infections. The younger the child, the less apt are the joints to be involved. In a large series the sole initial manifestations of rheumatic fever were growing and joint pains, chorea, and polyarthritis—each in about 25 per cent of cases. Active carditis was the sole manifestation in only 3 per cent.[210] In 15 per cent, active carditis was present at the onset.

In a study of 1,487 cases of rheumatic fever reported by Gibson[68] in Chicago, polyarthritis occurred in 983, chorea in 581; rheumatic nodules in 156; carditis in 864. Seventy-three and one-half per cent of the polyarthritis patients, 56 per cent of the chorea patients, 100 per cent of those having rheumatic nodules, and 84 per cent of the patients with carditis showed other rheumatic manifestations. Of 583 children with rheumatic heart disease, the initial manifestation was acute carditis in 8 per cent, polyarthritis in 53 9 per cent, chorea in 19.8 per cent, myalgia and joint pains in 9.6 per cent, and 8.7 per cent exhibited insidious carditis.[5] Pain in a joint of the lower extremity was the initial symptom in the majority of 100 cases studied by Kimbro[118] Sometimes this was so mild that the patients were referred in for flat feet. Dwyer[56] lists the most important symptoms as polyarthritis, carditis, fever, chorea, spontaneous nose bleed, pallor, anemia, and precordial, abdominal and pleural pains. In 1,754 necropsy examinations on rheumatic heart disease, a past history of articular involvement had been noted in only 29 per cent.[39] Shapiro[178] on the other hand obtained a positive history in all but 2 per cent of his cases. In a large series, Martin[133] failed to obtain a rheumatic history in only 14.7 per cent Hayes and Gibson[88] illustrate the importance of rheumatic nodules by pointing out that of 167 cases of rheumatic fever with nodules, every case presented some other manifestation of rheumatic fever. One hundred sixty-three of the 167 cases gave definite signs of heart disease. Supporting evidence was offered by Couper,[50] Banerjea,[10] and Struthers and Bacal[183]

Symptoms in children may include fatigue, malaise, growing pains, intermittent fever, tachycardia, pallor, anemia, night sweats, epistaxis, chorea, subcutaneous nodules, and cardiac murmurs. Epistaxis is frequently observed without trauma. It is usually controlled by cauteriza-

residual cardiac damage is not usually prevented nor remedied, though it is possible that it may be modified. Dr. May Wilson,[219] based on a study of 32 patients with active carditis expresses the opinion that the degree of residual cardiac damage is directly proportional to the duration of active carditis before treatment is instituted. The patients in this series were treated within the first few days and treated with ACTH for *seven days.* Massell[220] states: "There are hopeful indications that cardiac sequelæ may be altered provided treatment with the hormones can be started very early in the acute attack." He advises six to eight weeks of therapy.

An analysis of our cases and those reported by others indicates that there is a striking difference in the behavior of patients treated at the onset or within the first week of the disease as compared to those treated in later weeks or months. In the early treated cases clinical and laboratory signs of rheumatic activity usually subside rapidly and completely. Even more important is the finding that the early treated cases tend to relapse much

of rheumatic fever after a ten year follow-up was found to be 47 per cent and 20 per cent of the original thousand patients were dead.[107] Chronic rheumatic heart disease occurred in 25 per cent of children showing mild

cases early in hope of preventing permanent cardiac damage or determine to reserve these agents for life saving measures only in a grave carditis. Theoretically every acute attack of rheumatic fever should be treated early with cortisone or ACTH if prevention of sequelæ is the goal. There are, however, some other considerations to be weighed—some of them quite practical. All patients with rheumatic fever who are treated with ACTH or cortisone should be hospitalized for at least the first two weeks. The cost of ACTH and cortisone alone for the three weeks minimum treatment period is a factor. There are also some hazards and contraindications for their use which will be discussed shortly. Finally, sufficient time has not yet elapsed to be certain regarding the degree of protection from cardiac sequelæ even in early cases.

... of protection ... would ... where ... undue hardship and on those patients where carditis is severe.

Contraindications

ACTH or cortisone should not be administered to rheumatic fever patients in the presence of active tuberculosis, active peptic ulcer, diabetes,

tion; occasionally repeated epistaxis necessitates transfusion Joint pains and swelling may be entirely absent in children with rheumatic fever Rheumatic joint pains are more common in damp and rainy weather In children the pains are often relieved by lying in a warm bed, and may recur if the patient becomes cold. In some children the only evidence of the disease may be vague muscle pains, abdominal discomfort, fever, epistaxis, and a general feeling of sickness.[64]

In childhood the onset of rheumatic fever may be very difficult to recognize in the presence of respiratory infections. The affected joints may be somewhat painful, but there is little swelling Children often show no redness, swelling, or heat in the joints. Stiffness is more marked than tenderness, joints are only rarely hot or red.

Wilson found joint pains in 67 per cent of rheumatic children; in 26 per cent of cases, the joint pains were the first manifestation of the rheumatic process The most frequently involved joints in children were the ankles,

In children and young adults, rheumatic fever is often polycyclic with severe inflammatory reactions taking place in vascular tissues during each febrile phase of the cycle

In typical cases of rheumatic fever the diagnosis is relatively simple In children, the so-called "growing pains" may be a manifestation of this disease. These vary greatly but may consist of vague, dragging, fleeting pains in muscles and occasionally in joints, frequently accompanied by easy fatigability, loss of weight, and anorexia.

enough to produce a limp. Wilson has emphasized that growing and joint pains in childhood may be seen during infections (especially respiratory) or after excessive exercise or in association with foot strain, she has cautioned against interpreting all such pains as minor manifestations of rheumatic fever.[210]

Recent studies
(vague muscular
cluded that grow
they were not related to rheumatic fever.[87] Cardiac "rheumatism"

concluded that such leg pains, coming on characteristically at the end of the day and often awakening the patients during the night, were due to normal growth and not to rheumatic fever Others believe that "growing pains" are merely examples of foot strain in most instances

Cardiac Involvement.—The chief manifestation of rheumatic fever is carditis; the latter causes 40,000 deaths annually in this country. About one-third of patients with rheumatic fever develop active carditis, in the others, cardiac involvement remains subclinical. It may be difficult in some cases to ascertain whether the heart is affected (*i. e*, to an extent noticeable to the clinician), and whether transitory or permanent damage

from hormone treatment and sometimes acute exacerbations develop on withdrawal of the medication Advanced heart disease, with predominating mechanical failure and little or no active rheumatic fever, does poorly on this treatment because of the tendency to increased salt and water retention The presence of congestive heart failure due to active rheumatic carditis *is not a contraindication.* Patients with rheumatic heart disease

is doubtful and increased cerebral irritation may occur

Adverse Physiological Effects

The principal side effects noted have been round face, edema, insomnia, ulcer symptoms, acne, hirsutism, pigmentation, depression, weakness and tachycardia (late), hypertension, pulmonary edema and menstrual irregularities and vascular thrombosis The severity of the side effects appears to be related to the total amount of hormone received and the duration of time over which the hormone was received rather than upon the daily dose. The longer the hormone is administered the more noticeable and difficult to control are the side effects Very few serious side effects will occur in the three weeks' course of treatment if proper precautions as

with proper precautions, to warrant stopping treatment

Dosage and Routine of Administration

ACTH (corticotropin) should be given early in near maximum dosage averaging 80 to 100 I.U. daily Even small children tolerate 80 units daily and the dosage should not be calculated on a weight basis The total daily dose should be given intramuscularly in divided doses every six hours The initial maximum dosage is to be continued for seven days A reduction of 25 per cent in the dose is then made and continued for the next seven

of

at

terminate during the next seven days If, at the end of fourteen days of treatment, some evidences of carditis still remain, the dosage may be continued for an additional week without reduction and then gradually discontinued. It is, however, unlikely that if treatment was begun late and fourteen days of adequate treatment has not resulted in complete subsidence of carditis that further treatment will result in prevention of cardiac sequelæ. From the evidence available it appears quite likely that hope for the success of ACTH in preventing cardiac sequelæ depends upon very early and adequate treatment. There is no evidence to indicate that such

will result. At times this cannot be determined definitely for some months or years.[21] During the acute stage of the disease, signs of cardiac involvement include subjective symptoms (dyspnea, edema), the development of murmurs, or other adventitious cardiac sounds, disturbances in conduction of the heart (including electrocardiographic evidence) and changes in the size of the heart.

Marked dyspnea, peripheral edema, and signs of decompensation (including an enlarged liver) are uncommon during the acute stage of the disease, their presence would indicate cardiac damage. A prolonged elevation of temperature with a disproportionately high pulse rate is the rule and does not necessarily denote damage to the heart

he

ca

of

cardiac damage Too much stress has been placed upon cardiac murmurs and too little upon heart tones.[114] Systolic murmurs do not always indicate cardiac damage Soft systolic murmurs may be associated with fever,

In an occasional patient, electrocardiographic changes may be the only evidence that the illness is rheumatic A slight degree of delayed or disturbed conduction (which may not be evident clinically) can be detected by the electrocardiogram. Further delays of conduction may lead to actual dropping of ventricular beats

Another factor of importance during rheumatic fever is the size of the heart If the heart increases in size during an attack, it is probably damaged; however, the converse is by no means true. A normal-sized heart may be the seat of marked disease

A pericardial friction rub indicates cardiac involvement. If the peri-

unt of

Some individuals may continue throughout life without developing signs of rheumatic heart disease (unless a recurrence of rheumatic fever occurs)

The patient may develop a slight valvular lesion which is carried throughout life to a normal age expectancy (with proper care and the good fortune to escape a recurrence of the acute rheumatic attack)

Permanent heart damage results in at least one-third of these patients[123] The more frequent the recurrence of rheumatic fever, the greater is the likelihood of cardiac involvement The younger the patient, the more likely is the possibility of cardiac damage, this is apt to be more severe in young individuals

In rare instances (1 to 4 per cent of cases) death may occur during an acute attack of the disease from diffuse cardiac involvement (including auricular fibrillation and emboli)

cases treated late when carditis is well established will show reversal of long established carditis nor be prevented from developing cardiac sequelæ.

A Thorn test should be done at the beginning of treatment and it is also desirable to do a quantitative eosinophil count after forty-eight hours of treatment. *If the Thorn test does not show a definite drop in eosinophils* and they also remain at a high level after forty-eight hours of treatment cortisone should be substituted for ACTH. *If after one week of treatment with ACTH there has been no major improvement ACTH should be discontinued and the diagnosis reconsidered.* If nodule formation develops at the site of injection, maximum effect of the ACTH is not usually being obtained and it may be desirable to give the ACTH intravenously. This is done by dissolving 25 to 35 mg. of ACTH in 500 cc. of five per cent glucose in water. It is administered intravenously by the slow drip method requiring six to eight hours for administration. A more potent form of ACTH has recently become available and it is not deactivated by the muscle.

Cortisone May be Administered Intramuscularly or by the Use of Oral Tablets.—*The oral tablets should always be given in divided doses,* either one-half the daily dose every twelve hours or one-third the daily dose

[illegible]

water retention and indeed is vital where congestive failure is present or imminent. It is desirable when congestive failure is present to reduce sodium even lower until failure is relieved. The lowest practical diet is at a level of about 0.2 gram of sodium. It is well to point out that water softeners are usually operated by means of adding salt to the water. If water softeners are utilized, it is well to purchase distilled water for drinking and cooking

Potassium chloride must be administered when using these relatively high doses of ACTH and cortisone. In general 1 gram of potassium chloride may be given by dissolving soluble potassium chloride tablets in a glass of distilled water and taken after meals or it may be given in a syrup, or, if necessary, as enteric-coated tablets. Potassium chloride should not be given if there is evidence of renal involvement. The effects of hormonal

[illegible]

...e tend to mask infection and often prevent its localization. Also additional Group A hemolytic streptococcal infections may have been acquired since the precipitating infection The use of penicillin may prevent the spread of infection during treatment and may prevent additional absorption of the rheumatic fever producing substance produced by the Group A hemolytic streptococcus.

Rheumatic Fever Tends to Recur.—A patient who may have escaped valvular damage during his first attack may succumb to it in his second or third. Wilson believes that about 15 per cent of children escape some recurrent manifestation of activity of their rheumatic fever. She has also found a diminished tendency for recurrences starting at about puberty The most frequent form of recurrence is usually the same as the initial manifestation of the disease

The mitral and aortic valves are attacked most frequently. These may be involved separately or together Mitral insufficiency, alone, or in combination with aortic insufficiency may occur as early as forty-eight hours after the onset of an acute rheumatic attack [20] However, it requires months, and usually years, for sufficient scar tissue to be produced to result in valvular stenosis. This is important, inasmuch as stenosis of the valve ... fever.

85 per cent within a year of the onset of the rheumatic fever [210]

In children with rheumatic myocarditis, symptoms include fever (100° to ...

months).

In patients with active carditis, a marked decrease (30 or more per cent) of vital capacity may be seen With progressive cardiac involvement, one notes poor quality of the cardiac sounds, widely transmitted murmurs and possibly gallop rhythm In children, as the heart dilates with cardiac ...

edema, orthopnea, and a markedly engorged liver.

Subcutaneous Nodules.—These are usually seen in the subacute stage of rheumatic fever They may vary in size from a pin-head to an almond and are usually about the size of a split pea. Their most common location is about the elbows, fingers, knees, palms, neck, wrists, ankles, carpometacarpal joints, toes, occiput, or iliac crests They vary from 2 mm or less to more than a centimeter in width, and are usually discrete, but occasionally become conglomerate [210] Inasmuch as similar nodules may occasionally occur in rheumatoid arthritis, their presence is not pathognomonic of rheumatic fever

They are usually firm and painless and are not, as a rule, attached to the skin. They may rapidly increase in size and persist for several days, weeks, or months These nodules are noted more frequently in England than in this country

The frequency of such nodules is variable They have been reported in from 2 to 37 per cent of these patients.[4,8] The presence of nodules is considered to be indicative of a serious infection as they are often associated with severe and progressive cardiac damage

Bauer and his associates have carefully studied large numbers of these nodules [17] They attributed the pathologic changes in the nodules of rheumatic fever chiefly to injury of small blood-vessels, followed by an

The standard routine of rest, diet and general care as described elsewhere should be followed when ACTH or cortisone is utilized.

Salicylates should be given with the hormones and may be continued for a period after termination of hormone therapy

Length of Treatment

not be ideal, but it is predicated upon experience with early treatment and is, in our opinion, sufficient for the majority of early treated cases It is possible that if cases are treated within the first day or two of onset that seven days may be sufficient Most patients are not seen so early and the problem of correct diagnosis make this difficult As has been pointed out, the hope of prevention of cardiac sequelæ depends upon the earliest possible treatment. Following cessation of treatment there is frequently seen a short period of five to seven days of recurrent mild fever or increased sedimentation rate, and sometimes with occasional symptoms This has been called the rebound phenomenon The typical rebound phenomenon promptly subsides in not less than ten days without further treatment If the symptoms continue beyond seven to ten days, the disease is probably reactivated and may need further treatment The advantage of a short period of treatment (from fourteen to twenty-one days) is very real in that it avoids most of the problems of longer term administration of the hormones If these hormones interfere with the healing process, indeed it may [illegible] fore, it is [illegible] d carditis,

GENERAL TREATMENT OF RHEUMATIC FEVER

It should be obvious from the foregoing discussion regarding ACTH and cortisone that many patients with rheumatic fever are not suitable for ACTH or cortisone These patients must be treated with salicylates which still offer a very satisfactory method of treatment for the acute attack of rheumatic fever There are a few basic important methods of treatment that have stood the test of time and which are reviewed and tabulated here

Rest.—For the acute phase of rheumatic fever, complete rest is an indispensable agent, and it should be continued until all evidences of activity of the disease have disappeared These evidences of activity have been described elsewhere, but consist largely of joint pain, tachycardia, elevated sedimentation rate, leukocytosis with changes in the differential picture, fever, fatigue, anemia, etc. *The greatest possible rest,* which is desirable, [illegible]

Feiring,[164] who at an Air Forces convalescent center for rheumatic fever were impressed by the large number of cases who on arrival had disabling

exudation of plasma and blood cells into the connective tissue; small focal lesions similar to myocardial Aschoff nodules were also seen. Because of the predominantly exudative process in rheumatic fever, these nodules appear and resolve more quickly than those in rheumatoid arthritis.

Cutaneous Lesions.—Over 10 per cent of patients with acute rheumatic fever manifest cutaneous lesions; these are often overlooked.[210] They

by pale red or bluish-red rings, semicircles or polygons from 1 to 3 cm. in diameter. These are not raised but are macular, and do not result in scaling or pigmentation; there is no associated itching, swelling, or hemorrhage. The lesions are noted most often on the chest, abdomen, back, and thorax, and are uncommon on the extremities; they have not been reported on the face or in mucosae. Erythema annulare rheumaticum may be an initial symptom of rapidly fatal rheumatic fever.[1]

Rheumatic erythemas appear and disappear irregularly with wide fluctuations in intensity. Keil has presented a thorough review on rheumatic erythema.[113] The increased permeability of the capillary walls may be the pathologic basis for the skin lesions in rheumatic subjects. During a recent epidemic of rheumatic fever, crops of bluish, non-tender, maculo-

skin lesions: erythema nodosum, Osler node, psoriasis, scleroderma, dermatomyositis, systemic lupus erythematosus, and erythema diutinum[114]

The pathogenesis of **erythema nodosum** is open to question. Some workers have found a causal relationship between streptococcus hemolyticus infections and erythema nodosum, as well as between this skin lesion and rheumatic fever.[179] Others do not believe that erythema nodosum is

nanifesta-
vas rheu-
cases as

tuberculous when the tuberculin test was positive and as rheumatic when the test was negative

In a small school epidemic of erythema nodosum involving 14 cases, all of the boys were found to be highly sensitive to tuberculin, and 12 of these showed definite or suggestive lesions of tuberculosis.[25] Eight of the cases occurred in one cottage. A boy with open pulmonary tuberculosis had been removed from the school several weeks after the onset of the epidemic.

Wallgren[194, 195] and Van Henkelom[192] believe that erythema nodosum is a non-specific allergic skin eruption which appears especially in tuberculosis but occasionally in other infections (such as rheumatic fever).

Involvement of the Lungs and Pleura.—Rheumatic fever occasionally causes pleurisy and pneumonia. Pulmonary complications have been described, including bronchopneumonia, massive collapse of the lungs, pleural effusion, an

rheumatic pleurisy

pleural thickening

cent of children with other manifestations of rheumatic fever (as contrasted

anxiety neuroses. It was their deep conviction after personal interviews that the mental state of these patients was largely fostered by the preceding exhaustive therapeutic program which demanded absolute bed-rest throughout, and even beyond, the acute stage of rheumatic fever. They challenged the concept that confinement to bed implies the ultimate expression of rest *These authors define rest as maximum ease and comfort physically, mentally, and emotionally.* They studied 200 patients in this category and came to the conclusion that greater rest was actually secured in this phase of the disease when they were permitted to sit in or out of bed, and perhaps to walk a few steps to the latrine. They report that these

could not be drawn because the period of observation was not much over six months, but they earnestly request and invite attention to their suggestion for re-evaluation of the currently accepted policy of prolonged absolute bed-rest with its disadvantages in the treatment of rheumatic fever. At least we may be sure that rest, with whatever interpretation we may attach to it, is essential and constitutes the first essential anchor in the treatment of acute rheumatic fever

General Treatment of Rheumatic Fever

Rest in bed until signs of activity have disappeared, even after salicylates have been discontinued; this may require weeks, months or years [139]
Salicylates or aspirin in oral doses to tolerance (slight deafness and early ringing in ears is a satisfactory level). May vary widely from 5 to 15 gms daily
Local therapy to joints splinting, local analgesic applications
Removal of focal infection (especially tonsils) after the acute attack has subsided, there is no proof, however, that this lessens the severity of the cardiac lesion
Repeated small *transfusions* if severe anemia occurs, large amounts of iron if only moderately anemic
Adequate intake of calories and of all *vitamins*, but particularly of vitamin C (including 200 mg. of cevitamic acid orally each day in adults and half of this amount in children).
Sulfonamide and/or Penicillin prophylaxis *after the acute attack for several years to prevent recurrences* of hemolytic streptococcal infection and thereby rheumatic fever
Residence in an area of low incidence, if financially practical, at least until after puberty.

There
ose of
salicylates and method of administration. The majority of opinions, however, and the best studies (of which there have been several) reject the routine intravenous administration of salicylates as dangerous, toxic, and

with only 9 per cent in normal children). Others have found pleurisy to occur in from 2 to 20 per cent of patients with rheumatic fever, the hemor-

Pericarditis —Pericarditis has been reported to be much less common in rheumatic fever than myocarditis or endocarditis Acute pericarditis has been detected clinically in from 12 to 19 per cent of these patients [165] Rheumatic fever does not often cause extremely large pericardial effusions and paracentesis of these is practically never indicated Large doses of salicylates will usually lead to prompt relief and resorption of these effusions.[23]

A pericardial effusion in rheumatic fever may produce a chill with high

Pericardial involvement is often overlooked during life The physical signs of pericardial effusion will be present, including an enlarged area of

decreased as seen fluoroscopically.

Excluding tuberculous pericarditis, heart failure due to an adherent pericardium is usually the result of rheumatic infection [40]

COURSE, PROGNOSIS, AND END RESULTS OF RHEUMATIC FEVER

Recurrences are so numerous in rheumatic fever that they are almost the rule. The younger the patient, the more likely he is to have recurrences, and the higher is the mortality On the other hand, death rarely occurs during an acute attack of rheumatic fever, in a few cases, pancarditis, often associated with nephritis, leads to a fatal outcome in the acute phase of the disease. In at least one-third of the patients, permanent heart damage results

Exacerbations of the disease are frequently preceded by upper respiratory infections; however, other factors which may provoke attacks include

patients.

In a group of 54 patients in Boston, the disease was considered to be severe in one-third, moderately severe in one-half, and mild in one-fifth [77] The average duration of active infection in these patients was eight months, but 3 required bed-rest for two to two and one-half years. Of the 553

unnecessary. Oral administration can and does achieve equally satisfactory blood levels if such high levels are desirable. Among the advocates of high dosage, 35 mgs. per cent plasma level is considered an adequate level. There is, however, as yet no clear-cut evidence that administration of salicylates, even at toxic levels, will prevent the development and progression of rheumatic heart disease during the attack of acute rheumatic fever Most of the well-controlled studies[117,142,198,205] fail to demonstrate reduction in complications or sequelae with high doses, as compared to standard doses of salicylate This is a problem which cannot be settled now. The comparative value of large doses or smaller doses of salicylates will be determined only after a longer period of time has elapsed with follow-ups and

ates

to

er,

painful joints, etc. It is probable that the simple clinical observation of

prevents nausea, vomiting, and toxicity, is justifiable and worthwhile

Toxic reactions of salicylates, when given intravenously, are approximately 6 times as frequent even with the same blood level as when the salicylate is given orally

of vision, mild nausea, sl

evidences are not serious

with acute maniacal delirium These effects are almost never encountered with oral administration Very rarely with oral administration one may see, in addition to the mild symptoms of toxemia mentioned, shaking chills,

out evidence of intolerance If acute salicylate toxicity develops, treatment of choice is large quantities of intravenous lactate-Ringer's solution and dextrose

If salicylates are for some reason impossible to take orally, they may be given by rectal instillation, in starch paste, in approximately double the dosage given orally Absorption is not always adequate and results are not as satisfactory as when the drug is taken orally Salicylate treatment of whatever type should be continued until fever, articular inflammation, increased sedimentation rate, and other evidences of activity have disappeared. The question of rheumatic activity is a difficult one to be certain about Taran[194] believes that all the laboratory tests currently used in the diagnosis of rheumatic activity are inadequate in screening the presence or absence of activity He believes the most sensitive criterion for rheumatic activity is a careful study of the cardiac events in the electrocardiogram, particularly with relation to prolongation of electrical systole, the Q-T *time It is his opinion that this factor is the last one to return to* normal, and he believes this occurs perhaps long after temperature, pulse rate, blood count, sedimentation rate, hemoglobin, vital capacity, etc. have returned to normal

children followed by Ash[5] for an average of nine years, 71 per cent showed

more apt to cause a carditis in children than in adults.[6,111,116,163,178] DeLiee, Dodge, and McEwen[53] found only 7 per cent residual cardiac damage in

that 65 per cent were dead at the end of ten years. Cohn and Lingg[44] analyzed the records of 3,129 patients with rheumatic heart disease who died. Of this group with a fatal ending, the duration of the disease was about thirteen years. Fifty per cent died within nine years after onset, 25 per cent lived more than seventeen years, and 10 per cent for thirty years or more. Whereas Cohn and Lingg analyzed only fatal cases, Martin[133] *studied a group of 1,438 children with rheumatic heart disease;* 30 per cent of this group were dead after twenty years and 42.6 per cent of these deaths occurred within the first five years of the initial rheumatic

matic fever, found that at the end of ten years 313 or 31.3 per cent had no *detectable heart disease, 470 or 47 per cent had some degree of rheumatic* heart disease, 203 or 20.3 per cent had died, and most of them as the result of recurrence of rheumatic fever; and in 14 or 1.4 per cent the follow-up data were inadequate Of the 1,000 patients 648 or 64 8 per cent are able to lead relatively normal lives ten years after the onset of their rheumatic fever. Jucá and White[109] analyzed 100 unselected fatal cases of rheumatic heart disease over the age of twenty years at death. It was *found that congestive failure without evident acute rheumatism accounted* for 35 per cent of the fatalities, acute rheumatic fever and congestive failure for 22 per cent, bacterial endocarditis for 18 per cent, other causes related to the heart for 14 per cent, causes unrelated to the heart for 8 per cent, acute rheumatic fever alone 2 per cent, and unknown causes 1 per

...s of
with
ritis

cent of the rheumatic fever, 6 per cent of the arthritis, and 2 per cent of the chorea cases were dead at the end of six years. Cotton,[49] in a study of 200 cases of rheumatic fever under observation for ten years, reports that 29 per cent of mitral disease with systolic murmur, 46 per cent of mitral stenosis, 50 per cent of aortic regurgitation and mitral stenosis, and 30 per cent of aortic regurgitation were dead at the end of ten years. Wilson and Lubschez[213] *in studying 499 rheumatic individuals during 5,677 person*

Other Drugs.—These may be summarized very briefly. *Sulfonamides are not helpful during the acute attack,* though penicillin may be used to eradicate Group A streptococcus infection during the first week. Aminopyrine, cinchophen and neocinchophen, colloidal charcoal injections, the calcium double salt of benzoic acid and succinic acid benzol esters, and various other drugs have been reported with some success. However, none have been proven superior, nor stood the test of time with salicylates.

Additional Therapy for the Patient With Acute Rheumatic Fever Includes:

1. *Good nursing care.*
2. *Rest in bed* during the acute stages of the disease.
3. *Relief of cardiac pain* by an ice-bag to the precordium, or by codeine or morphine if needed
4. Liquid or semisolid *diet* in the acute stages
5. *Fluids* should be *forced* to replace water lost by sweating
6. Cooling baths to reduce fever are unnecessary because salicylates are so effective, and local applications for the joint phenomena are usually unnecessary.
7. *Soft pillows* may be used *as splints* under and around inflamed joints in the acute stages
8. *Aspiration of pericardial effusion* is *seldom required* but may be needed occasionally if the amount of fluid is large enough to interfere with cardiac function; thoracentesis may be needed if pleural effusion produces *circulatory or respiratory difficulty.*
9. If congestive heart failure occurs, use low-sodium diet (200 mgs.); dilute hydrochloric acid or ammonium chloride; xanthine or mercurial diuretics, no rigid fluid restriction; and lastly, if carditis is acute and severe ACTH or cortisone
10. [illegible] 0.015 to [illegible]

    lent manifestations occur, *careful nursing* is required to guard against personal bodily injury from the uncontrolled movements and *fever therapy* should be given (aiming to produce a temperature of from 104° to 105° F. for three to five hours; the fever therapy must be repeated for an average of six sessions, as needed). Milder forms of chorea which do not respond to conservative care may require fever therapy.

Treatment of Congestive Failure During Active Rheumatic Fever.—While digitalis may always be used in the presence of congestive heart failure, children with active rheumatic fever do not respond uniformly well to the use of digitalis. It is now considered good practice to use all other methods of restoring fluid and electrolyte balance before resorting to the use of digitalis in the presence of an active inflamed myocardium. A low- [illegible] Distilled [illegible] e installed. [illegible] ammonium [illegible] mercurial [illegible] ive failure is due to active carditis and does not respond to simple measur[illegible], ACTH or

Cortisone should be administered for control of the carditis In grave carditis these may be life saving. Walsh and Sprague[295] found xanthine

Transition From Bed Rest to Activity in Patients With Rheumatic Fever:[117]

1. When the sedimentation rate has returned to or nearly to normal, and when the rectal temperature, pulse and leucocyte count have remained normal for ten to fourteen days in the absence of antirheumatic drugs and when there is no evidence of joint pain, subcutaneous nodules or

2 *Walking on the level* is permitted next and finally stair climbing, if necessary
3 *Rest in bed must be resumed temporarily if signs of increased rheumatic activity appear at any time.*
4. *Mild sports* (but no strenuous competitive activity) *are permitted and may be encouraged in individuals without evidence of cardiac embarrassment. Too close restriction may lead to the development of cardiac neuroses.*

States.
6 Statistical studies show *no evidence that eradication of foci of infection favorably affects the course of rheumatic fever* Some believe that tonsillectomy is dangerous if done early in the acute stage of rheumatic fever In rheumatic individuals who are susceptible to sore throats and tonsillitis, it is usually agreed that tonsils should be removed during an inactive stage of the disease

Local Therapy to the Joints.—The swollen and painful joints may be protected by pillow splints, or molded plaster, castex, or lucite Relief following salicylates is usually so prompt that splints are seldom required If the lower limbs are involved, a cradle should be placed over the legs so

CHAPTER 33

PALINDROMIC RHEUMATISM

By Edward F. Rosenberg, M.D.

The term "palindromic," derived from Greek roots meaning "to recur," was employed by Hippocrates to characterize erysipelas and other ailments tending to appear repeatedly in the same individual This term has

General Features.—Palindromic rheumatism occurs infrequently The number of cases reported up to the present numbers approximately 100 Only 34 cases were found by careful search among a series of many thousands of rheumatism sufferers who were examined for this condition between the years 1928 (when palindromic rheumatism was first recognized) and 1940 (when the first publication on the subject appeared). Since 1940 sporadic papers have been published concerned with palindromic rheumatism, confirming its existence and describing individual cases or small series. These papers have not unfolded the mysterious etiology of palindromic rheumatism, neither has knowledge regarding pathology and treatment been notably advanced since the original description

Each attack is followed by a complete remission, and most attacks run their courses within a few hours to a day or two Only in rare instances have attacks persisted as long as a week

Practically every joint is liable to the attacks However, a distinct predilection is noted for finger joints. Other favored sites are dorsum of the hand, knees, wrists, shoulders, ankles, elbows, temporomandibular, sternoclavicular joints, and cervical vertebrae

Onset of attacks may occur at any time of day but there is a distinct tendency for attacks to appear in the late afternoon In general, the attacks begin with great suddenness, often patients describe the onset as "instantaneous" Within a few minutes, an affected joint may become painfully swollen, maximum severity may be reached within a few hours after the moment of onset Pain is variable, usually moderate but sometimes quite severe Some patients feel a steady throbbing ache, others experience dull and persistent discomfort as after a bruise In some pain is violent and may be described as "bursting" or a feeling as though the joint were gripped in iron tongs

and not infrequently is sufficient to iron out the wrinkles, making the skin tense or waxy

five days.[127] In very occasional cases, even with local splinting of the involved joints, codeine or morphine may be required.

Tonsillectomy.—If the tonsils are definitely infected, they should be removed *after the acute infection has subsided.*

Swift believed that tonsillectomy seemed indicated if the tonsils remained diseased because of the frequency with which rheumatic fever followed tonsillitis.[184] He believed that the tonsils might be the site of production of sensitizing substances or areas of infection from which bacteria were discharged into the blood stream. However, the removal of tonsils is not absolute insurance against recurrences of the disease inasmuch as patients may have other sources of sensitization or may incur reinfection with hemolytic streptococci.

Kaiser summarized the effect of tonsillectomy in 48,000 rheumatic children,[110] and concluded that: "Tonsillectomy is justified in practically every rheumatic child until other factors that influence this disease are better understood "

(1) Tonsillectomized children are as likely to develop muscular rheumatism and chorea.

(2) Tonsillectomized children are as likely to develop subsequent rheu-[illegible]

[illegible]

(4) The mortality was nearly twice as high in children with tonsils at the initial attack as among those tonsillectomized before the initial attack.

PREVENTION OF RHEUMATIC FEVER

Prevention or prophylaxis of rheumatic fever warrants the utmost emphasis as it offers us the only vulnerable point of attack in controlling this disease, which, to repeat, is the leading fatal disease from the age of five through eighteen Despite the fact that as yet we have no single specific etiological agent nor any single specific cure, there are some proven facts already described in regard to the etiology of rheumatic fever that warrant our increased efforts and vigorous attack toward its prevention.

Prevention of rheumatic fever must be directed first toward preventing streptococcal infections, and, secondly by early treatment of streptococcal infections in the hope of reducing the amount of unknown substance produced by the streptococcus which causes the rheumatic fever.

Prevention of Hemolytic Streptococcal Infections

Sulfonamide Prophylaxis.—Mass prophylaxis with sulfonamides was first used in our armed forces during World War II Probably nearly two million men at some time received prophylactic sulfonamide. In most instances the drug used was sulfadiazine, in 1 gram daily doses. Many carefully controlled studies were done on comparable groups containing several thousands of troops.[12,43,97,100,197,214] Reduction of respiratory and streptococcic diseases and rheumatic fever on the treated groups was striking, varying from 50 to 85 per cent. Toxic effects were mild and infrequent. Successful mass prophylaxis for an epidemic, however, depends on the organism being sulfa-sensitive.

In palindromic rheumatism, osteoporosis does not appear and destructive lesions are not to be found.

Although in both classic and so-called "infective" varieties of rheumatoid arthritis, patients may have short preliminary bouts of arthritis with more or less complete remissions, these attacks are apt to last weeks or months, not a few hours or a day or two, as in palindromic rheumatism.

Palindromic rheumatism differs notably from periarticular fibrositis in that episodic swellings, redness and severe tenderness, all outstanding features of attacks of palindromic rheumatism, are not found in fibrositis.

Intermittent hydrarthrosis, a rare remitting form of arthritis, affects the knee joints in every instance. Attacks of intermittent hydrarthrosis are usually painless and the attacks are regularly spaced. In contrast, attacks of palindromic rheumatism occur at irregular intervals, affect many joints other than knees, and generally, are moderately or severely painful

Rheumatic fever may be distinguished from palindromic rheumatism in [illegible] febrile, last for weeks or months

[illegible] from gout by absence of hyperuricemia, subcutaneous tophi and progressive roentgenographic destruction of affected joints. Patients with palindromic rheumatism are not relieved by a regimen which, in most instances, promptly relieves attacks of gout. Attacks of palindromic rheumatism are shorter and occur more frequently than is the case with most attacks of gout.

A disease designated angioneural arthrosis and described in 1911 by Solis-Cohen,[17] seemingly has some features in common with palindromic [illegible] is, however, has not been described in [illegible] consequently it is difficult to assess its [illegible] iatism. Features of the condition as described by Solis-Cohen included attacks of painful articular swellings associated with discolorations and fever. Episodes often persisted for many [illegible] Hives [illegible]

[illegible] affected." In palindromic rheumatism, fever has not been observed; most attacks have lasted but a few hours or a day or two, and the majority of attacks are nonarticular. Hives and dermographism were not a feature of the cases, the great majority of which were in adults.

The term [illegible] ally in this country, was employed [illegible] articular rheumatism characterized [illegible] involving articular or [illegible] distant from joints. [illegible] resembled palindromic rheumatism but there were important [illegible] Occurrence of fever and erysipeloid rashes, high incidence of allergic reactions, and absence of redness, characterized the disease. These features are not to be found in palindromic rheumatism.

Treatment.—Many remedies have been tried for palindromic rheumatism, including measures directed against allergy, psychic disturbances, and infections. Histamine, epinephrine, ephedrine, benzedrine and newer anti-allergic drugs including benadryl and pyribenzamine have been tried

Individual Prophylaxis —The use of sulfonamide for the prevention of recurrent rheumatic fever attacks has been studied since 1936 when Thomas and her co-workers, and Coburn and Moore, began their studies From the literature[158] and personal communications, we have tabulated reports from 13 groups of investigators who have carried out control studies with sulfonamide prophylaxis. If the experience of all investigators is summarized, the following information is obtained —Rheumatic subjects were given sulfa prophylaxis for a total of 1280, person seasons (person season equals 1 person for 1 season). There were 30 recurrent attacks of rheumatic fever in this group, or 2 3 per cent The control groups not receiving sulfa prophylaxis total 1,630 person seasons with 228 (13 9 per cent) recurrent attacks It seems fairly clear that those not receiving sulfonamide prophylaxis had 6 times as many recurrent attacks of rheumatic fever as those on the prophylaxis.

Toxic reactions were in general rare and usually mild They consisted usually of rashes, drug fever, or leukopenia Unless toxic reactions are manifest in the first month, they are not likely to occur The early studies were largely confined to the use of the drug during the winter months Recently considerable support has developed for the use of the prophylaxis throughout the year

Penicillin Prophylaxis —No very large groups have as yet been studied with penicillin prophylaxis but a number of reports have indicated its

return from school instead of at noontime

There are three possible disadvantages to oral penicillin prophylaxis·

1. Oral penicillin may be responsible for developing a hypersensitivity to penicillin This is not common, but when it occurs that individual is deprived of his most potent weapon if acute streptococcal infection develops.
2. Development of penicillin-resistant organisms other than Group A may occur Fortunately the degree of resistance is usually not great and large doses of penicillin, if needed, are generally effective
3. The year around cost of penicillin prophylaxis is very high compared with the cost of sulfadiazine

The same indications serve for penicillin prophylaxis as for sulfa prophylaxis Only known rheumatic individuals need to use prophylaxis regularly. Short period prophylaxis with sulfa or penicillin may be used by any one

without success. Measures directed against a supposed infection included removal of foci of infection, administration of sulfanilamide and typhoid vaccine shock therapy. None of these succeeded. Various patients reported relief from miscellaneous measures but it was not possible to be sure that the improvement noted was related to the treatments. Thus patients claimed relief after calcium gluconate and measures of psychotherapy, such as vacations and adopting children

Three patients with palindromic rheumatism have been treated successfully with soluble gold compounds by Boland and Headley.[1] In each instance, a complete arrest of articular manifestations resulted. To the authors, this suggested that palindromic rheumatism may be a variant of rheumatoid arthritis.

No specific data have thus far been published concerning treatment of palindromic rheumatism by means of adrenal or pituitary hormones. Although scattered cases have apparently achieved symptomatic remission on these hormones, it is doubtful that they should be used as treatment except as a desperation measure for this mild form of rheumatic disease.

Prognosis.—Among patients observed prior to 1944, the outcome was a spontaneous cure in 15 per cent, and reduction in severity in 44 per cent. The disease had remained unchanged in 25 per cent and in 11 per cent was aggravated

Death had resulted in only one instance and in this case the cause of death was unrelated to the palindromic rheumatism No tendency was noted for attacks to become confluent and none of the patients had developed chronic articular changes

Subsequent Data.—Case reports describing additional patients with palindromic rheumatism have appeared irregularly during the years since this disease was first called to the attention of physicians. Patients with palindromic rheumatism have been observed throughout the United States[3,4,6,9,11,13,14,16,18,20,21] and also in several overseas countries.[4,12,19,22,23]

matism be enlarged to include intermittent hydrarthrosis, Ménière's syndrome, and certain cases of iritis Ropes and Bauer[15] considered palin-

toms of rheumatoid arthritis

It would seem, however, that little can be gained by including other diseases of unknown cause under the designation of palindromic rheumatism In all likelihood however, the study of this group of patients will be facilitated by carefully distinguishing them from other recognizable

the distinctive features In palindromic rheumatism, repeated attacks affect different joints, para-arthritis and periarthritis occur frequently, attacks often affect finger pads. In "episodic" rheumatoid arthritis,

vapors (aerosols) also gave encouraging results. Newer developments include air purifiers that, by means of electrical charges, precipitate particles of dust, pollens, bacteria, etc. from the air. While these various methods of controlling air-borne infections are not as yet very practical, they offer considerable promise in this direction for the future.

Climate Prophylaxis.—Meteorological factors unquestionably play a rôle in the frequency of initial attacks and recurrences of rheumatic fever. This influence of geography and climate has been reported under predisposing factors. These studies have been tabulated under comparison of hospital admission rates for rheumatic fever, comparable necropsy figures, and comparison of the incidence of rheumatic fever among comparable groups These studies show that rheumatic fever and rheumatic heart disease are 10 to 20 times as common in some northern geographical areas as in other southern ones.

During the past twenty years Holbrook and Hill[102] have followed more than 300 rheumatic fever cases transferred to the Southwest desert with a minimum length of observation of two years and a maximum of eighteen ... approximately 2.0 per cent

period of five years.

It is fairly obvious from the above facts that climate and meteorology play an important rôle in the prevention of this disease. We do not as yet know what the specific meteorological factors are that are essential, and unfortunately a selective artificial climate is not yet practical. It should also be clear that despite the favorable influence of climate, a very small number of the many thousands needing protection from this disease can afford to use climate as a preventive measure.

Prevention of Rheumatic Fever by Early Treatment of Streptococcal Infection

Until very recently there was not sufficient evidence that the incidence of rheumatic fever could be altered after the streptococcal infection had been initiated Earlier studies by Massell have now been confirmed by a study from an Air Force Base, Code 219, by Capt. Lewis W. Wannamaker, M.C., *et al.*,[221] indicating that adequate *penicillin treatment in an early streptococcic infection will strikingly reduce the incidence of* subsequent ... at Fort Warren of 1,177 patients ... o developed ... 28 developed ... n-rheumatic patients was 1,200,000 units intramuscularly during the first ninety-six hours Rheumatic individuals—that is, those with previous rheumatic fever or with rheumatic heart disease—should be treated for at least eight days with 300,000 units daily intramuscularly, or three times that amount orally.

This method is no substitute for prophylaxis in the rheumatic individual because only about one-half the streptococcal infections causing rheumatic fever are recognizable clinically. Clinical indications for the use of peni-

In palindromic rheumatism, osteoporosis does not appear and destructive lesions are not to be found

Although in both classic and so-called "infective" varieties of rheumatoid arthritis, patients may have short preliminary bouts of arthritis with more or less complete remissions, these attacks are apt to last weeks *or months*, not a few hours or a day or two, as in palindromic rheumatism

Palindromic rheumatism differs notably from periarticular fibrositis in that episodic swellings, redness and severe tenderness, all outstanding features of attacks of palindromic rheumatism, are not found in fibrositis.

Intermittent hydrarthrosis, *a rare remitting form of arthritis, affects the* knee joints in every instance. Attacks of intermittent hydrarthrosis are ... attacks ... joints

Rheumatic fever may be distinguished from palindromic rheumatism in that attacks of rheumatic fever are usually febrile, last for weeks or months and often are associated with carditis.

Palindromic rheumatism is distinguished from gout by absence of hyperuricemia, subcutaneous tophi and progressive roentgenographic destruction of affected joints Patients with palindromic rheumatism are not relieved by a regimen which, in most instances, promptly relieves attacks of gout. Attacks of palindromic rheumatism are shorter and occur more frequently than is the case with most attacks of gout.

A disease designated angioneural arthrosis and described in 1911 by Solis-Cohen,[17] seemingly has some features in common with palindromic rheumatism. Angioneural arthrosis, however, has not been described in further publications since 1911 and consequently it is difficult to assess its relationship to palindromic rheumatism Features of the condition as described by Solis-Cohen included attacks of painful articular swellings ... Episodes often persisted for many

attacks have lasted but a few hours or a day or two, and the majority of ... were not a feature of

...ally in this country, ...rticular rheumatism characterized by recurring episodes of local edema, involving articular or periarticular tissues, tendons or subcutaneous tissues distant from joints This disease, like angioneural arthrosis, has not been identified definitely in this country. In some respects, the condition described by Kahlmeter ... important differences

Treatment.—Many remedies have been tried for palindromic rheumatism, including measures directed against allergy, psychic disturbances, and infections Histamine, epinephrine, ephedrine, benzedrine and newer anti-allergic drugs including benadryl and pyribenzamine have been tried

cillin treatment are the presence of exudative lesions on the tonsils or in the throat and/or a respiratory infection with a temperature of 101° or over. For purposes of treatment, such patients may be assumed to have a streptococcal infection However, failure to respond to penicillin therapy within thirty-six to forty-eight hours would indicate that no hemolytic streptococcal infection was present.

[illegible]

suspected too strongly of heart disease and condemned needlessly to years of limitation of activity and a deep-seated cardiac neurosis

Despite the fact that we still know very little of the exact mechanism producing rheumatic fever, it is evident that a full-scale and vigorous application of the information we now have would materially reduce the incidence and recurrences of this disease.

Summary of Prevention

The incidence and hazards of rheumatic fever and rheumatic heart [illegible]

[illegible] protein, eggs, and vitamins

(3) The heating and ventilating systems of schools, movies, theatres, and homes should be modernized and newer and practical methods for the control of air-borne infections installed as soon as they become available.

(4) Serious consideration should be given to the advisability of administering sulfonamide and/or penicillin prophylaxis to every *rheumatic subject living in an area of high incidence in an attempt* to prevent recurrences Experience now seems to indicate that approximately 5 out of 6 recurrences can be prevented by this method Multiple effective antibiotics may soon be available at [illegible]

(5) [illegible] rheumatic fever

(6) Climate prophylaxis may be used when necessary and available, with a high degree of efficacy

without success. Measures directed against a supposed infection included removal of foci of infection, administration of sulfanilamide and typhoid vaccine shock therapy. None of these succeeded. Various patients reported relief from miscellaneous measures but it was not possible to be sure that the improvement noted was related to the treatments. Thus patients

authors, thus suggested that palindromic rheumatism may be a variant of rheumatoid arthritis.

No specific data have thus far been published concerning treatment of palindromic rheumatism by means of adrenal or pituitary hormones.

The disease had remained unchanged in 25 per cent and in 11 per cent was aggravated

Death had resulted in only one instance and in this case the cause of death was unrelated to the palindromic rheumatism No tendency was noted for attacks to become confluent and none of the patients had developed chronic articular changes

Subsequent Data—Case reports describing additional patients with palindromic rheumatism have appeared irregularly during the years since this disease was first called to the attention of physicians. Patients with palindromic rheumatism have been observed throughout the United States[2,3,6,9,11,12,14,16,18,20,21] and also in several overseas countries.[4,12,19,22,23] The enlarging scope of these observations has served to disclose the widespread occurrence of this syndrome and has produced various speculations as to its relationship to rheumatoid arthritis, emotional ailments, angioneurotic edema, etc One physician described the condition among children.[16] Another writer recommended that the scope of palindromic rheumatism be enlarged to include intermittent hydrarthrosis, Ménière's syndrome, and certain cases of iritis. Ropes and Bauer[15] considered palindromic rheumatism to represent an aberrant or atypical form of rheumatoid arthritis, pointing out that discrete episodes of articular pain and swelling such as occur in palindromic rheumatism may be observed as early symptoms of rheumatoid arthritis

It would seem, however, that little can be gained by including other diseases of unknown cause under the designation of palindromic rheu-

affect different joints, para-arthritis and periarthritis occur frequently, attacks often affect finger pads. In "episodic" rheumatoid arthritis,

BIBLIOGRAPHY

1. Abramson, H , and Tunick, A. M., Jour. Pediat , 13, 94, 1938
2. Apfel, Harry: Arch Pediat , 61, 409, 1944
3. Aschoff, L Verhandl. d deutsch path Gesellsch, 8, 46, 1904.
4. Ash, Rachel: Am Jour. Dis. Child , 52, 280, 1936.
5. ———: Am Heart Jour., 22, 439, 1941.
6. ———: Pennsylvania Med. Jour., 44, 484, 1941.
7. ———: Am Jour Dis Child , 63, 1, 1942.
8. Med , 67, 241, 1941; Proc
9.
10.
11.
12. Bassett, Major A. M · Use of sulfadiazine as respiratory disease prophylaxis in the Eighth Air Force, News Letter (AAF Rheumatic Fever Control Program), 2, 4-7, 1945
13. Bauer, E L . Pennsylvania Med. Jour., 49, 113, 1945.
14. Baxter, E H , Hartwell, R M , and Peck, L E.· Jour Am Med Assn , 111, 2476, 1938
15. Bell, E T Am Jour Path , 8, 639, 1932
16. Omaha Mid-West Clin Soc , 2, 59,
17. 940
18.
19.
20.
21.
22.
23.
24.
25.
26. Health
27.
28.
29.
30.
31. Jour , 95, 234, 1942
32. Cahan, J M · Pennsylvania Med Jour., 44, 481, 1941.
33. ——— · Med Rec , 153, 441, 1941
34. Chandler, C A., and Taussig, H B Bull Johns Hopkins Hosp , 72, 42, 1943
35. Chávez Ignacio Am Heart Jour., 24, 88, 1942
36. Dis Child 48, 69, 1934.
37.
38.
39. 4, 684, 1941.
40.
41. Coburn, A. F 1942
42. ——— Am Jour. Dis Child , 65, 744, 1943
43. Coburn, A F. Jour Am Med Assoc , 124, 88, 1944
44. Med Assoc 71, 1-8; 113-117, 1943
45.
46.
47.
48.
49.
50. Couper, E C R Arch. Dis Child
51. Davis, D. B , and Rosin, Sidney. Jour Pediat , 24, 502, 1944
52. Decherd, G M , Jr , and Herrmann, G. R., Texas State Jour Med , 39, 229, 1943.

In palindromic rheumatism, osteoporosis does not appear and destructive lesions are not to be found.

Although in both classic and so-called "infective" varieties of rheumatoid arthritis, patients may have short preliminary bouts of arthritis with more or less complete remissions, these attacks are apt to last weeks or months, not a few hours or a day or two, as in palindromic rheumatism.

Palindromic rheumatism differs notably from periarticular fibrositis in that episodic swellings, redness and severe tenderness, all outstanding features of attacks of palindromic rheumatism, are not found in fibrositis.

Intermittent hydrarthrosis, a rare remitting form of arthritis, affects the knee joints in every instance. Attacks of intermittent hydrarthrosis are usually painless and the attacks are regularly spaced. In contrast, attacks [illegible]

[illegible] that attacks of rheumatic fever are usually febrile, last for weeks or months and often are associated with carditis.

Palindromic rheumatism is distinguished from gout by absence of hyperuricemia, subcutaneous tophi and progressive roentgenographic destruction of affected joints. Patients with palindromic rheumatism are not relieved by a regimen which, in most instances, promptly relieves attacks of gout. Attacks of palindromic rheumatism are shorter and occur more frequently than is the case with most attacks of gout.

A disease designated angioneural arthrosis and described in 1911 by Solis-Cohen,[17] seemingly has some features in common with palindromic rheumatism. Angioneural arthrosis, however, has not been described in further publications since 1911 and consequently it is difficult to assess its relationship to palindromic rheumatism Features of the condition as described by Solis-Cohen included attacks of painful articular swellings associated with discolorations and fever. Episodes often persisted for many [illegible] of the cases affected multiple articulations. Hives [illegible]

affected. In palindromic rheumatism, fever has not been observed; most attacks have lasted but a few hours or a day or two, and the majority of attacks are nonarticular. Hives and dermographism were not a feature of *the cases, the great majority of which were in adults.*

The term "allergic rheumatism," regarded skeptically in this country, was employed by Kahlmeter[10] to describe a form of articular rheumatism characterized by recurring episodes of local edema, involving articular or periarticular tissues, tendons or subcutaneous tissues distant from joints *This disease, like angioneural arthrosis, has not been identified definitely* in this country. In some respects, the condition described by Kahlmeter [illegible] but there were important differences [illegible] e of allergic reactions, and [illegible] These features *are not to be found in palindromic rheumatism.*

Treatment.—Many remedies have been tried for palindromic rheumatism, including measures directed against allergy, psychic disturbances, and infections Histamine, epinephrine, ephedrine, benzedrine and newer anti-allergic drugs including benadryl and pyribenzamine have been tried

53 DELIEE, ELVIRA M, DODGE, KATHARINE G, and MCEWEN, CURRIER Am. Heart Jour, 26, 681, 1943
54 DEVENHOLZ, E J, and RAMBAR, A C Am Jour Dis Child, 61, 1044, 1941
55 DUBLIN, W B · Dis Nerv System, 2, 390, 1941
56. DWYER, HUGH, L Proc. Inter-State Postgrad Med Assembly of North Amer, Oct 1943.
57 EHLERTSEN, C F. Acta med Scandinav., 62, 353, 1942
58 EPSTEIN, E Z, and GREENSPAN, E B Arch Int Med, 68, 1074, 1941
59 EPSTEIN, N, and MCDONALD, F G · New England Jour Med, 222, 943, 1940
60 ERNSTENE, A C, and SCHNEIDER, R W · Am Jour Med Sci, 202, 737, 1941
61 FAHR, T. in Handbuch d spez path Anat u Histol (Henke and Lubarsch), Berlin, Julius Springer, 6, Pt 1, 121, 1925
62 FASHENA, GLADYS J.. Texas State Jour Med, 40, 278, 1944
63 ——— · Texas State Jour Med, 39, 474, 1944
64 FEASBY, W. R War Med, 5, 216, 1944
65 FOSTER, D B Arch Neurol and Psychiat, 47, 254, 1942
66 FOSTER, J L Penna Med Jour, 44, 476, 1941
67. GELFMAN, RAYMOND Ann Int Med, 19, 253, 1943
68 GIBSON, STANLEY Jour Tennessee Med Assoc, 34, 219, 1941
69 GLAZEBROOK, A J, and THOMSON, SCOTT Edinburgh Med Jour, 48, 674, 1941
70 GLOVER, J A Lancet, 2, 51, 1943
71 GRAY, J W, FENDRICK, E, and GOWEN, C H Texas State Jour Med. 28, 202, 1932
72 GREEN, C A Med Press, 205, 523, 1941
73 ——— Jour. Path and Bact, 53, 223, 1941
74. ——— Jour. Hyg, 42, 365, 371 and 380, 1942
75 ——— Ann. Rheumat Dis, 3, 4, 1942
76 GRIFFITH, GEO C, and HUNTINGTON, ROBERT W, JR Ann Int. Med, 25, 283, 1946
77 GRIFFITH, GEO C, MOORE, FREDERICK J, MCGINN, SYLVESTER, and COSBY, RICHARD S Am Heart Jour, 35, 447, 1948
78 GRISHMAN, A., and KANE, I J Am Jour Roentgenol, 53, 575, 1945
79 GUY, P F Northwest Med, 43, 166, 1944
80 HADFIELD, G, MAGEE, V, and PERRY, C B Lancet, 1, 834, 1934
81 HALL, E M, and ANDERSON, LUCILLE R. Am Heart Jour, 25, 64, 1943
82 HAMILTON, B E, and THOMSON, K J The heart in pregnancy and the childbearing age, Boston, Little, Brown & Co, 1941.
83 HANSEN, A E Jour Am Med Assoc, 121, 987, 1943.
84 ——— Journal-Lancet, 63, 138, 1943
85 HARDGROVE, M, WHITTIER, L, and SMITH, E R Jour Am Med Assn., 130, 488, 1946
86 HARTWELL, A S Proc Staff Meet Clin., Honolulu, 11, 17, 1945
87 HAWKSLEY, J C Jour Royal Inst Pub Health and Hyg, 1, 798, 1938
88 HAYES, R M, and GIBSON, STANLEY Jour Am Med Assoc, 69, 554, 1942
89 HEDLEY, O F Public Health Reports, No 1983, 55, 1707, 1940
90 ——— Pub Health Bull, 268, 1, 1941.
91 ——— Reprint No 2195, United States Public Health Service, Washington, D C, 1941
92 HEFFER, E. T Jour Pediat, 18, 363, 1941
93 HENCH, P. S Ann Int Med, 12, 1005, 1939
94 HENCH, P S, *et al* Ann Int Med., 14, 1383, 1941.
95 [illegible]
100 ——— Jour Am Med Assoc, 126, 81, 1944
101 HOLBROOK, W P, and STEPHENS, C A L JR. Tucson, Arizona, Unpublished data, 1948
102 HOLBROOK, W P Prevention of Rheumatic Fever, address delivered at Annual Clin Conference, Chicago Med Society, March 5, 1948
103 HUBBLE, DOUGLAS Brit Med Jour, 1, 121, 154, 1943

without success. Measures directed against a supposed infection included removal of foci of infection, administration of sulfanilamide and typhoid vaccine shock therapy. None of these succeeded. Various patients reported relief from miscellaneous measures but it was not possible to be sure that the improvement noted was related to the treatments Thus patients claimed relief after calcium gluconate and measures of psychotherapy, such as vacations and adopting children.

Three patients with palindromic rheumatism have been treated successfully with soluble gold compounds by Boland and Headley.[1] In each instance, a complete arrest of articular manifestations resulted To the authors, this suggested that palindromic rheumatism may be a variant of rheumatoid arthritis

No specific data have thus far been published concerning treatment of palindromic rheumatism by means of adrenal or pituitary hormones. Although scattered cases have apparently achieved symptomatic remission on these hormones, it is doubtful that they should be used as treatment except as a desperation measure for this mild form of rheumatic disease.

Prognosis.—Among patients observed prior to 1944, the outcome was a spontaneous cure in 15 per cent, and reduction in severity in 44 per cent. The disease had remained unchanged in 25 per cent and in 11 per cent was aggravated

Death had resulted in only one instance and in this case the cause of death was unrelated to the palindromic rheumatism No tendency was noted for attacks to become confluent and none of the patients had developed chronic articular changes

Subsequent Data.—Case reports describing additional patients with palindromic rheumatism have appeared irregularly during the years since this disease was first called to the attention of physicians. Patients with palindromic rheumatism have been observed throughout the United States[2,3,6,9,11,13,14,16,19,20,21] and also in several overseas countries[4,17,19,22,23]

matism be enlarged to include intermittent hydrarthrosis, Ménière's syndrome, and certain cases of iritis Ropes and Bauer[14] considered palin-

toms of rheumatoid arthritis

It would seem, however, that little can be gained by including other diseases of unknown cause under the designation of palindromic rheumatism In all likelihood however, the study of this group of patients will be facilitated by carefully distinguishing them from other recognizable

affect different joints; para-arthritis and periarthritis occur frequently, attacks often affect finger pads. In "episodic" rheumatoid arthritis,

104 HUTCHESON, W. C.: Arch. Path, 31, 369, 1941.
105 HUTTON, R. LER, and BROWN, C. R Ann Int Med, 20, 85, 1944.
106 JENNINGS, C. G, SHOTT, KATHLEEN, V, and MARTIN, A. G.: *The relative frequency of respiratory diseases and rheumatic fever at Kerns, Utah*, News Letter, 2, 6, 1945.
107. JONES, T. D., a
108 JONES, T. D.:
109 JUCÁ, ANTONIO,
110 KAISER, A. D.
111 KEEFER, C. S: Clinics, 1, 117, 1942
112 KEIL, H: Ann Int. Med, 10, 1686, 1937.
113 KEIL, H.: Medicine, 17, 261, 1938.
114 KEIL, H.: Ann. Int. Med, 11, 2223, 1938
115 KEITH, J D.: Canad. Pub Health Jour., 32, 95, 1941.
116 ———: Canad Med Assoc. Jour., 45, 119, 1941.
117 KEITH, J. D, and ROSS, ALAN: Canad. Med. Assoc. Jour., 52, 544, 1945
118 KIMBRO, R. W.: Texas State Jour Med, 41, 296, 1945.
119 KING, J. T. Ann Int Med, 16, 462, 1942
120
121
122
123
124
125.
126
127
128

129
130.
1029, 1943
131 LICHTY, J. A, JR.: Am Jour. Dis Child, 62, 92, 1941.
132. LONGCOPE, W. T.. *Am Jour Med Sci*, *152*, *625*, *1916*
133 MARTIN, A. T. Jour. Am. Med Assoc, 117, 1663, 1941.
134 MASSELL, B F., and JONES, T. D *Am Heart Jour*, *27*, *575*, *1944*.
135 MASTER, A M: Am Heart Jour., 27, 634, 1944.
136 MCEWEN, C, ALEXANDER, R C, and BUNIM, J. J.. Jour Lab. and Clin. Med, 21, 453, 1936
137 MCEWEN, O C. Bull New York Acad Med, 19, 679, 1943.
138. METROPOLITAN LIFE INSURANCE COMPANY: Mortality among children with

139. — 24, 9, 1943.
140 —
141 MOTE, J. R, and JONES, T. D: Jour Immunol, 41
142. MURPHY, G E.: Bull Johns Hopkins Hosp, 77, 1, 1945.
143. MYERS, W. K, KEEFER, C. S, and HOLMES, W. F, JR: Jour Clin. Invest, 14, 119 1935
144 OFFENKRANTZ, F. M: Am. Jour Dis Child, 56, 67, 1938.
145 OSLER, W: On Chorea and Choreiform Affections, Philadelphia, P. Blakiston & Sons, 1894
146 health
147 politan

148
149.
150
151. Med,
152

153.
154

In palindromic rheumatism, osteoporosis does not appear and destructive lesions are not to be found.

Although in both classic and so-called "infective" varieties of rheumatoid arthritis, patients may have short preliminary bouts of arthritis with more or less complete remissions, these attacks are apt to last weeks or months, not a few hours or a day or two, as in palindromic rheumatism.

Palindromic rheumatism differs notably from periarticular fibrositis in that episodic swellings, redness and severe tenderness, all outstanding features of attacks of palindromic rheumatism, are not found in fibrositis

Intermittent hydrarthrosis, a rare remitting form of arthritis, affects the knee joints in every instance. Attacks of intermittent hydrarthrosis are usually painless and the attacks are regularly spaced In contrast, attacks of palindromic rheumatism occur at irregular intervals, affect many joints other than knees, and generally, are moderately or severely painful.

Rheumatic fever may be distinguished from palindromic rheumatism in that attacks of rheumatic fever are usually febrile, last for weeks or months and often are associated with carditis.

Palindromic rheumatism is [illegible]
hyperuricemia, subcutaneous [illegible]
destruction of affected joints. [illegible]
not relieved by a regimen which, in most instances, promptly relieves attacks of gout. *Attacks of palindromic rheumatism are shorter* and occur more frequently than is the case with most attacks of gout.

A disease designated angioneural arthrosis and described in 1911 by Solis-Cohen,[17] seemingly has some features in common with palindromic [illegible] -is, *however*, has *not* been described in [illegible] consequently it is difficult to assess its [illegible] natism Features of the condition as described by Solis-Cohen included attacks of painful articular swellings *associated with discolorations and fever. Episodes often persisted* for many [illegible] ives, [illegible] ents [illegible] ften [illegible] nost
attacks have lasted but a few hours or a day or two, and the majority of attacks are [illegible] were not a feature of the cases, [illegible]

The ter [illegible] *y in this country*, was emplo [illegible] icular rheumatism characterized by recurring episodes of local edema, involving articular or periarticular tissues, tendons or subcutaneous tissues distant from joints *This disease, like angioneural arthrosis*, has not been identified definitely in this country. In some respects, the condition described by Kahlmeter resembled palindro [illegible] differences
Occurrence of fever [illegible]
tions, and absence [illegible]

[illegible] or palindromic rheuma- [illegible] sychic disturbances, and [illegible] benzedrine and newer anti-allergic drugs including benadryl and pyribenzamine have been tried

155 Reitersbach, G , and Kuttner, A. G : Am Heart Jour , 20, 573, 1940
156 Reitersbach, G , Lenert, T. F , and Kuttner, A G . Jour Clin Invest , 20, 289, 1941
... Air

... Med , Chicago, 15, 270,

163. Riecker, H. H Jour Michigan Med Soc , 40, 208, 1941
164 Rinehart, J F.: Ann Rheumat Dis , 3, 154, 1943
165 Ritchie, W. T.· Brit Med Jour , 1, 679, 1936
166 Robertson, H. F , Schmidt, R E , and Feiring, Wm Am Jour Med Sci , 211, 67-73, Jan., 1946
167 Robinson, J. J · Arch Pediat , 61, 561, 1944
168 ——— · Arch Pediat , 61, 6, 1944
169. ——— *Ibid* , 62, 387, 1945
170 Robinson, R W · Am Heart Jour , 29, 378, 1945
171 Rosenberg, E F , Baggenstoss, A H , and Hench, P S Ann Int Med , 20, 903, 1944.
... Heart Jour , 23, 71, 1942

... nd Shearer, Margery C

175 Schwartzman, J , Dragutsky, D , and Rook, G Jour Pediat , 19, 201, 1941
176 Shambaugh, P Arch Int Med , 50, 921, 1932
177. Shank, R E , Coburn, A F , Moore, Lucile, V , and Hoagland, C L Jour Clin Invest , 23, 289, 1944

... U S Nav Med Bull , 43, 1, 1944

... Am Med Assn , 114, 629, 1940
... d Med Assoc Jour , 46, 376, 1942
... heme, 5th ed , Philadelphia, W B

... elen, N Jour Immunol , 49, 209,

186 Taran, L M : Jour Pediat , 24, 62, 1941
187 Taran, L M , Jablon, J. M , and Weyr, Helen N Jour Immunol , 51, 53, 1945
188 Taran, L M Am Jour Med , 2, 368, 1947
189
190
191
192
193
194
195
196
197
198
199
200
201
202 Watson, R. F., Rothbard, Sidney, and Swift, H F Jour Am. Med Assoc , 128, 1145, 1945

CHAPTER 34

INTERMITTENT HYDRARTHROSIS

By Charles Ragan, M.D.

This is a chronic disorder characterized by periodic recurrences of joint

involvement In certain instances this syndrome may be the initial symptom of definite rheumatoid arthritis which subsequently develops [16]

Since the first report[26] in 1845, various reviews (Benda, Berger, Bierring, Linberger, Schlesinger) have appeared The disease is relatively uncommon in the literature since only about 120 cases have been reported. We have seen 6 such cases in three years and doubtless many cases have not been recorded Five of these patients have been followed for five years and none have developed evidence of rheumatoid arthritis.

Etiology.—This is unknown Cases have occurred in individuals between the ages of seven and sixty, most often between the ages of twenty and forty-five Early reports are open to question as many of these represented other forms of arthritis In our experience, the disease has a definite prefer-

mented upon include trauma,[12 23] *infection with Brucella abortus,*[1 24 25]

any more than suggestive evidence as to the etiologic agent and the opinion that the etiology is unknown remains accepted The analogy of inter-

interval between attacks - seven to eleven days (limits three to twenty-one days, only rarely beyond these). (4) Duration of effusion—three to five days. (5) Absence of pronounced local heat, muscular spasm, notable tenderness or severe constitutional reaction (6) Local disability, decreased motion and occasionally, marked pain (7) Total duration of cycles of disease: up to twenty-two years

In our experience, most cases of intermittent hydrarthrosis begin in the second decade. Although the disturbance may be bilateral, in a majority of cases only one knee is involved Other joints (as the ankle, hip and rarely a joint of the upper extremity) are occasionally attacked

203 WEDUM, A G., and WEDUM, BERNICE G.· *Cincinnati Jour. Med*, 23, 489, 1942
204. ———. Am Jour. Dis. Child, 67, 182, 1944
205. WÉGRIA, RENÉ, and SMULL, KATHARINE Jour. Am Med Assoc, 129, 485, 1945
206 WEINSTEIN, JOSEPH Ann Int Med, 21, 252, 1944
207 WEISS, M. M Am Heart Jour, 22, 112, 1941.
208 WHEELER, S M., and INGRAHAM, H S U S. Nav. Med. Bull, 45, 253, 1945
209 WHITING, W B.: Medical Papers, Christian Birthday, Baltimore, Waverly Press, p. 125, 1936.
210. WILSON, M. G · Rheumatic fever, New York, The Commonwealth Fund, 1940
211 WILSON, MAY G, SCHWEITZER, M D, and LUBSCHEZ, ROSE. Pediatrics, 22, 468, 581, 1943.
212. WILSON, MAY G Jour Am Med Assoc., 124, 1188, 1944
213. WILSON, MAY G, and LUBSCHEZ, ROSE· Jour Am Med Assoc, 126, 477, 1944
214 [illegible]
215 [illegible]
216 [illegible]
217 [illegible]
218. [illegible]
219. [illegible]
220 [illegible]
221. [illegible] 673, 1951.
222 QUINN, R W Jour. Clin. Invest, 27, 471, 1948
223 [illegible] 217, 174, 1949
224 [illegible] 3, 482, 1949
225 [illegible], 1949
226. [illegible] LLANDER, J L · Ann

attacks tend to affect the same joints again and again, persisting for longer periods than attacks of palindromic rheumatism. Often, days or even weeks pass before an attack of episodic rheumatoid arthritis subsides, in contrast [illegible]

[illegible]

patients with episodic rheumatoid arthritis generally reveals persistence of at least some of the rheumatic symptoms between attacks, whereas the palindromic pa[illegible]

Between attack[illegible]
toms, disturbed lal[illegible]
swelling and tenderness are apt to be present. In such instances there is often slight but persisting loss of weight, excessive fatigue, vasomotor disturbances, anemia, and an elevated sedimentation rate indicating that all is not well even though joints may be nearly or entirely free of symptoms Roentgenograms may show early signs of osseous or articular damage in episodic rheumatoid arthritis whereas the roentgenogram reveals little of interest in palindromic rheumatism

The problem of palindromic rheumatism is not a large one but this curious and distinctive disease stands as a challenge to clinicians who should learn to recognize it and to be prepared to explain correctly to patients its distinctively benign character. Palindromic rheumatism poses a challenge also to researchers who have yet to elucidate the nature of this ailment, its cause and proper treatment.

BIBLIOGRAPHY

1. Boland, E W., and Headley, N. E.· Ann Rheum Dis, 7, 246, 1948
2 Cain, J. C. Jour Am Med Assn, 125, 1037, 1944
3. Ferry, J L Jour Indiana Med Assn, 36, 348, 1943.
4 Forestier, J · Revue du rheumatisme, 13, 78, 1946
5. Francon, F Revue du rheumatisme, 13, 17, 1946
6 Grego, J G, and Harkins, H. N : Jour Michigan Med. Soc, 43, 401, 1944
7. Hench, P S, and Rosenberg, E. F : Arch Int Med, 73, 293, 1944 Proc. Staff Meet, Mayo Clinic, 16, 808, 1941
8 Hench, P S, *et al* (Ninth Rheumatism Review), Ann. Int Med, 28, 66, 309, 1947.
[illegible], 454, 1947.

[illegible] 14, 101, 1944
14 Paul, W D, and Carr, T L · Arch Physical Med, 26, 687, 1945
15 Ropes, M. W, and Bauer, W. New England Jour Med, 233, 592, 618, 1945
16 Salomon, M I New York State Jour Med, 46, 622, 1946
17 Solis-Cohen, S: Trans. Coll Phys, Philadelphia, 33, 309, 1911; Trans. Assn [illegible]
18 Th[illegible]
19. Va[illegible]
20 Va[illegible]
21 — [illegible]
22 Weber, F. P Lancet, 2, 931, 1946.
23 Wingfield, Alec: Brit Med. Jour, 2, 157, 1945

CHAPTER 34

INTERMITTENT HYDRARTHROSIS

By CHARLES RAGAN, M.D.

THIS is a chronic disorder characterized by periodic recurrences of joint

involvement. In certain instances this syndrome may be the initial symptom of definite rheumatoid arthritis which subsequently develops.[16]

Since the first report[25] in 1815, various reviews (Benda, Berger, Bierring, Linberger, Schlesinger) have appeared The disease is relatively uncommon in the literature since only about 120 cases have been reported. We have *seen 6 such cases in three years and doubtless many cases have not been* recorded. Five of these patients have been followed for five years and none have developed evidence of rheumatoid arthritis

Etiology.—This is unknown. Cases have occurred in individuals between the ages of seven and sixty, most often between the ages of twenty and forty-five Early reports are open to question as many of these represented other forms of arthritis In our experience, the disease has a definite prefer-

marlaria, rheumatic fever, lymphogranuloma, psychic factors,[9] sensitization to food,[19 29 34] bacteria or other agents,[6,22] association with menses,[33] childbirth,[6 10 23] and endocrine disturbance.[14] In none of these has there been

interval between attacks—seven to eleven days (limits three to twenty-one days, only rarely beyond these) (4) Duration of effusion—three to five days (5) Absence of pronounced local heat, muscular spasm, notable tenderness or severe constitutional reaction (6) Local disability, decreased motion and occasionally, marked pain (7) Total duration of cycles of disease up to twenty-two years

In our experience, most cases of intermittent hydrarthrosis begin in the second decade Although the disturbance may be bilateral, in a majority of cases only one knee is involved Other joints (as the ankle, hip and rarely a joint of the upper extremity) are occasionally attacked

matism were listed as such in the British series, whereas psychoneuroses were responsible for joint and muscle symptoms in 1 out of every 5 rheu-

that non-articular rheumatism associated with the psychoneuroses has not been given the prominence which it deserves

COMMON SITES OF FIBROSITIS

The areas most commonly affected by fibrositis are the neck, shoulder and scapular areas, the lower back, gluteal region and the chest wall. The soft tissues of the face, hands and feet are seldom involved Some have stressed the frequent involvement of abdominal muscles, but pain of undetermined origin in the abdomen should not be "disposed of" as fibrositis of abdominal muscles until more is known of this condition[22]

ETIOLOGY

As mentioned before, no single etiological factor has been established As in the case of other rheumatic conditions, there seem to be many precipitating or aggravating features which have the support of tradition and experience

Infection.—The direct infective theory originally proposed by Stockman now has little support He believed the lesion to be the result of bacterial invasion, but was unable to demonstrate any organism Nevertheless the occurrence of articular and non-articular rheumatism in association with influenza, malaria, dysentery, brucellosis and other general infections is often observed

must be considered an experimental procedure based on poor theoretical grounds

common in bus drivers, miners, policemen, gardeners, stokers and milkmen.

patients with episodic rheumatoid arthritis generally reveals persistence of at least some of the rheumatic symptoms between attacks, whereas the palindromic patient is entirely well when his attacks subside.

Between attacks of episodic rheumatoid arthritis, constitutional symptoms, disturbed laboratory findings, and sometimes slight residual articular swelling and tenderness are apt to be present. In such instances there is often slight but persisting loss of weight, excessive fatigue, vasomotor disturbances, anemia, and an elevated sedimentation rate indicating that all is not well even though joints may be nearly or entirely free of symptoms. Roentgenograms may show early signs of osseous or articular damage in episodic rheumatoid arthritis whereas the roentgenogram reveals little of interest in palindromic rheumatism.

The problem of palindromic rheumatism is not a large one but this curious and distinctive disease stands as a challenge to clinicians who should learn to recognize it and to be prepared to explain correctly to patients its distinctively benign character. Palindromic rheumatism poses a challenge also to researchers who have yet to elucidate the nature of this ailment, its cause and proper treatment.

BIBLIOGRAPHY

1. Boland, E W, and Headley, N E. Ann Rheum Dis, 7, 246, 1948
2. Cain, J C Jour Am Med Assn., 125, 1037, 1944
3. Ferry, J L Jour Indiana Med Assn, 36, 348, 1943
4. Forestier, J Revue du rheumatisme, 13, 78, 1946
5. Francon, F Revue du rheumatisme, 13, 17, 1946
6. Grego, J G, and Harkins, H N · Jour. Michigan Med Soc, 43, 401, 1944
7. Hench, P S., and Rosenberg, E. F. Arch Int Med, 73, 293, 1944 Proc Staff Meet, Mayo Clinic, 16, 808, 1941.
8. Hench, P S, *et al* (Ninth Rheumatism Review), Ann Int Med, 28, 66, 309, 1947.
9. Hopkins, J J, and Richmond, J B. Ann Int Med, 26, 454, 1947.
10. Kahlmeter, G Acta med. Scandinav., 102, 432, 1939
11. Mazer, Milton Jour. Am Med Assn, 120, 364, 1942.
12. Neligan, A R. Brit Med Jour, 1, 205, 1946
13. Pa [illegible]
14. Pa [illegible] 945
15. Ro [illegible]
16. Sa [illegible] Assn
17. So [illegible]
18. Th [illegible]
19. Va [illegible]
20. Va [illegible]
21. ———. Ann Int Med, 19, 122, 1943
22. Weber, F P.: Lancet, 2, 931, 1946.
23. Wingfield, Alec Brit Med Jour., 2, 157, 1945

Unilateral fibrositis of the shoulder muscles was described in a group of London women forced to st[illegible] long hours during the war.[11] [illegible] strains may be the importa[illegible] Fibrositis.

Exposure.—There would seem to be no doubt that exposure to cold, [illegible] *temperature or weather* can initiate an [illegible] body, perhaps previously overheated is [illegible] history. Some susceptible individuals even find it unwise to work in the cooler, damper atmosphere of the basement in their home and such people assiduously avoid air-conditioned rooms, which seem to provoke symptoms in various body sites

Psychological Factors.—In discussing etiology, there remains no doubt in the mind of the experienced observer, that the symptom complex of *fibrositis may be initiated or profoundly affected by the emotional state* of the individual. The trend of modern medicine to consider the patient as a "whole" (mind and body) rather than the body alone has revealed that there may be a physical as well as a mental reaction in emotional states and that muscles often participate in this reaction with resultant aches and pains and evidence of tension. The observant physician cannot help but be impressed by the high percentage of patients with this syndrome who show evidence of a tension state.

Halliday[20] considers that in most cases, fibrositis is a psychosomatic disorder, the symptoms being an outward manifestation of inner emotional [illegible] or internal [illegible] esent the [illegible] this way [illegible] ttention [illegible] is bodily [illegible] when the [illegible]

In a study of the psychosomatic aspects of chronic non-a[illegible] matism Ellman and Shaw[18] noted that the term fibrositis is applied to many *maladies sharing a common symptomatology* of pain, stiffness, and limitation of movement. [illegible] patients over a period of four years [illegible] iefly by exclusion, [illegible] gross nature of the symptoms and the poverty of the physical findings They point out that muscles serve as means of attack and defense in the struggle for existence and thus internal tension is most easily relieved by muscular action.

Following this study Ellman and Shaw state "it seems clear that the nature of this often very prolonged incapacity is psychiatric in the majority of cases. The rheum[illegible] is merely part of the neurotic disor[illegible] ct he [illegible] rheu- aches in his mind."

CHAPTER 34

INTERMITTENT HYDRARTHROSIS

By Charles Ragan, M.D.

This is a chronic disorder characterized by periodic recurrences of joint

involvement In certain instances this syndrome may be the initial symptom of definite rheumatoid arthritis which subsequently develops [16]

Since the first report[26] in 1845, various reviews (Benda, Berger, Bierring, Linberger, Schlesinger) have appeared The disease is relatively uncommon in the literature since only about 120 cases have been reported. We have seen 6 such cases in three years and doubtless many cases have not been recorded Five of these patients have been followed for five years and none have developed evidence of rheumatoid arthritis

Etiology.—This is unknown Cases have occurred in individuals between the ages of seven and sixty, most often between the ages of twenty and forty-five Early reports are open to question as many of these represented other forms of arthritis In our experience, the disease has a definite prefer-

interval between attacks—seven to eleven days (limits three to twenty-one days, only rarely beyond these) (4) Duration of effusion—three to five days (5) Absence of pronounced local heat, muscular spasm, notable tenderness or severe constitutional reaction (6) Local disability, decreased motion and occasionally, marked pain. (7) Total duration of cycles of disease. up to twenty-two years

In our experience, most cases of intermittent hydrarthrosis begin in the second decade Although the disturbance may be bilateral, in a majority of cases only one knee is involved Other joints (as the ankle, hip and rarely a joint of the upper extremity) are occasionally attacked.

matism" because it perpetuates the idea that rheumatism is actually present. They point out that if the patient has an anxiety state, it should be diagnosed as such and thus the road is opened to more positive therapy where the role played by tension, and the need for adjustment in the particular environmental situation may be explained.

Weiss[38] also deprecates the frequent diagnosis of fibrositis and early arthritis for the many patients who complain of aches and pains in muscles and joints associated with chronic fatigue.

In a study of 83 patients with the symptom complex of fibrositis, previously labelled with fifteen different diagnoses, psychosomatic study proved the presence of psychopathology rather than tissue pathology. Hysteria was encountered in 37 patients, anxiety states in 22, hypochondriasis in 4, and 2 suffered from psychotic depressions Eighteen patients could be classified as having neurotic character disorders. Psychologic symptoms most frequently encountered were poor sleep and poor sexual

In another study, Walters, Slade and Graham[37] point out that the fibro-

stretched to include most of the average emotional trouble in this unhappy world. They could however be termed emotionally unstable since they reacted with excessive emotional responses both mentally and physically When they met their particular trials of life they became "tense," "keyed-up," "pent-up," "tied in knots," "held in," "unable to relax," *etc.* A

These authors contend that *no sharp line can be drawn between psychogenic rheumatism and the fibrositis syndrome.* In psychoneurotic patients who are

There is usually an abrupt onset of an increasing effusion in a knee with moderate pain and limitation of motion. There are no marked signs of local inflammation. Within several days, the fluid slowly disappears and the knee assumes a normal appearance. After a variable period of time, which is often fixed for each individual, joint effusion reappears, this is usually from seven to twenty-one days. The leukocyte count remains normal in most instances.



FIG 198 —Intermittent hydrarthrosis of the knees (see case report)

In true intermittent hydrarthrosis, the sedimentation rate is normal, there is no anemia and there is no evidence of a generalized disease such as fever, anorexia, weight loss or muscle wasting. In the presence of any of these signs or symptoms, a good prognosis such as is the rule with intermittent hydrarthrosis, cannot be given and another cause such as rheumatoid arthritis or rheumatic fever must be entertained.

Recurrence of the disease may continue for years, and even after long periods of remission. A well-known story is that of the athletic schedule of a prominent university which was especially arranged (supposedly) to permit matches to fall upon days when a stellar athlete, who suffered from intermittent hydrarthrosis, knew he was to have a remission.

Psychogenic Rheumatism, and Fibrositis respectively. Yet, a careful review of their content reveals that all three groups are discussing the same clinical state This would seem to add further proof that multiplicity of terms has led to confusion and a diagnosis based on etiology would do much to clarify this situation.

The role of psychological factors in the fibrositis syndrome, however, has not been universally accepted. The skepticism of the proponents for organic origin of the disease was expressed by Kelly[25,26] who challenged the psychosomatic theory and stated "the symptoms can be explained on the basis of known somatic reactions rather than of mystic symbolism and emotional delusion."

The exact role of psychogenic factors in the etiology of fibrositis, therefore, remains controversial but to most observers the syndrome is most commonly seen in nervous and anxious people who appear to have a "tension state" and who find it difficult or impossible to relax.

Miscellaneous Factors.—Many other causal factors have been suggested, *among them metabolic and endocrine disturbances, postural defects, cir-*
cu

e
wc is
may be caused by early and reversible chemical changes in the connective tissue which might either persist without progression, or which could eventually progress to the pathologic states of the Collagen Diseases, if the unknown cause is of sufficient virulence. This reasoning is so far purely hypothetical, but further knowledge of the histochemistry of connective tissue may settle this point.

Trigger Areas or Myalgic Spots.—*Any discussion as to the actual* cause of these tender, so-called trigger areas is still largely speculative. Although many believe that trauma plays a large part in the development of myalgic spots or nodules, the exact etiology and pathology is still unknown

and that such pain has a physico-chemical origin. It was postulated that *fibrositis is due to some local disturbance of metabolism*, arising in different cases from a variety of factors including trauma, infection, vascular disturbances, etc.

Kelly[25] feels that nervous reflexes are instrumental in the causation of fibrositic lesions. His theory is also based on the work of Lewis, 1942, on referred pain and tenderness He believes that traumatic, infective, and other conditions may give rise to reflex effects of muscle spasm, deep tenderness and even tissue changes through alteration of circulation and through a direct effect on the vital function of cells. The myalgic spot itself is a small, tender area, but it may give rise to a large referred area of secondary tenderness These painful areas, therefore, represent reflex phenomena which would *explain the lack of tissue change in many* biopsies which have been taken.

Copeman[16] feels that the myalgic spot is the early lesion and that the presence of a palpable nodule is a sign of *chronicity* *In an* interesting study during an epidemic of influenza, lumbar and other pains were a prominent feature, the acute lumbar pain being associated with localized

Typical Case Résumé.—A typical history in one of our patients, a laboratory technician, was as follows.

At about thirteen years of age the left knee began to swell at irregular intervals *X*-rays of the knee and joint aspiration were done and she was advised to use

vaccine was made from a tooth socket and given in conjunction with diathermy with remission in the intermittent hydrarthrosis after about a month.

At twenty, the left knee again began to swell An autogenous vaccine from the throat was used on this occasion The swelling recurred at fairly regular intervals of twelve days during the course of the injections but ceased soon after the injections were stopped

In February 1946, at age twenty-six, swelling of the left knee again began and recurred regularly at eleven to twelve day intervals This continued for a year

persisted until the summer of 1949 when the amount of swelling gradually decreased and, since 1950, no hydrarthrosis has appeared At no time during this course has the patient felt any ill effects, save some tightness in the knees in the presence of fluid with inability to flex them fully When fluid is absent, motion is complete without pain She has been able to continue working throughout and during our observation of her, she has never shown fever, leukocytosis or an elevated sedimentation rate The hyaluronic acid of the joint fluid removed on numerous occasions has had the characteristics of normal joint fluid hyaluronic acid *X*-rays of knees have been negative Swelling recurred while on pyribenzamine and ephedrine, and while on elimination diets

Differential Diagnosis.—This is usually not difficult *Other causes of*

synovitis[28]

Findings have varied in the few cases studied to date. In one, the synovial surface was covered with long slender branching villi with edematous tissue and profuse infiltration with lymphocytes in both villi and

tender areas in the lumbar muscles (myalgic spots). On re-examination of these patients one and two months later these spots were still present, but in diminishing numbers. The patient was usually not aware of this until pressure from the examining finger disclosed them. These patients did not have a previous rheumatic history. Copeman believed that myalgic spots could arise in the course of acute influenza and that some of these

of the presence of such spots until they may become activated by trauma, chilling or systemic infection.

Gordon[18,19] considers the fibrositic nodule to be the hallmark of the fibrositic subject, but the tombstone of the fibrositic attack.

Elliott[13,13] has warned that the interpretation of tenderness as a physical sign may lead to certain pitfalls. The local tender areas in fibrositis do not necessarily represent a local lesion. He pointed out that the myalgia in muscles may arise from an irritated nerve root and such a mechanism may resemble fibrositis not only clinically, but in its response to procaine injection. He reports 3 cases of local tenderness over the sacroiliac area, 1 from an epidural abscess at lumbar three, another due to extrusion of the fourth lumbar disk, and the third due to tuberculosis of the sacroiliac joint. He believes that such tender spots are due to local muscle spasm from an irritated nerve root, and electromyographic studies were said to support this. The presence of a central cause for local muscle tenderness must therefore always be considered.

PATHOLOGY

performed in large numbers, but the pathological picture remains vague and ill-defined. Stockman[35] described the pathology of fibrositis as an

proliferation of fibroblasts, appearance of numerous minute new blood vessels and absence of polymorphoneuclear leukocytes in the exudate. There was no formation of pus. The resultant swelling was usually soft, small and ill-defined. These inflamed excrescences may then resolve spontaneously or from therapy, or more often increase in size because of the proliferation of connective tissue cells so that the mass became tough and fibrous.

The larger of these lesions are said to be the size of an almond and can be felt as ill-defined swellings along the edges of muscles or in subcutaneous tissue and fascia. These new formations may be scattered all over the body in large numbers or may be confined to one or a few parts, much of this changed tissue is too minute to be detected except by its tenderness on pressure.

subsynovial surface. Dense connective tissue, fat and sclerotic vessels were also found.
cell infiltration wa
thickening of the
observed patholog
matoid arthritis[27] In one of our patients a synovectomy was performed

surface These changes are indistinguishable from those described for rheumatoid arthritis, yet this patient had involvement only of the knees and had a normal sedimentation rate Swelling of one or other knee continued for one year and then with no treatment recurrence stopped. She has been followed now for one and a half years without symptoms or x-ray evidence of disease and residual consists of minimal synovial thickening of knees by palpation. Function is complete. The synovial fluid may contain several thousand cells per c.mm. with more than 50 per cent of polys.[7] The synovial fluid polysaccharide—hyaluronic acid—in the cases of this syndrome which we have examined, appears to be normal. This would

joints of patients with active rheumatoid arthritis. This observation is in keeping with the concept that intermittent hydrarthrosis is a local tissue response and not a generalized disease such as is rheumatoid arthritis

regularly for years in many instances without change in the nature of the symptoms.

Treatment.—Little success has been obtained with older forms of therapy

ous typhoid vaccine, ergotamine tartrate[8,36,37] and local roentgen therapy.[21] Pregnancy has also had an ameliorating effect upon several cases.[13]

The periodicity of intermittent hydrarthrosis has suggested a possible allergic basis Service has reported 4 cases with allergic histories in which the joint symptoms were relieved by correcting the existing allergy. He feels that *food allergy* is an important factor in hydrarthrosis. Others reported cases due to food sensitization. One of these was due to ingestion fish from the

Krida, one patient had attacks for nine months coming on five weeks after trauma and persisting despite conservative therapy, no positive skin tests were found although careful examination for sensitivity was performed In the second case, the etiology seemed to be related to the menses

Slocumb[33] described 3 stages of the process, (1) a low grade inflammatory serofibrinous exudate with proliferation of fibroblasts and blood vessels, (2) local tissue thickenings or *gross subcutaneous nodules*, and (3) fibrous

than is normal. This will mean an increase in tension on activity, more *pain on movement*, and either a greater *liability to further acute attacks* of fibrositis or the setting up of a state of chronic fibrositis when, owing to thickening, shortening and tension of fibrous tissue, all movement becomes *painful*. A *vicious circle* has been set up, and any subfascial fat is in an increased state of tension."

These theories, however, still await histological confirmation.

Steinberg[34] feels that fibrositis is a metabolic disorder and that vitamin E is necessary for the prevention and cure of the particular connective tissue change. He claims the pathology is similar to that of nutritional muscular dystrophy in young rats which responds to the administration of alpha tocopherol. This view has, as yet, been unconfirmed.

A pathological basis for pain and local tenderness in certain cases has been adequately established by Copeman and Ackerman.[8] The pain is related topographically to the basic fat pattern and was ascribed to edema in the lobules and the development of tension as they swell and fill their compartments This may lead to herniation of the fatty tissue if the fascial walls are partially deficient Such fat hernias were demonstrated in 20 cases at biopsy and their removal gave persistent relief. Histological studies revealed no cellular infiltration so that some process other than inflammation had to be assumed.

The occurrence of such fat herniations has now been proven by many others and it becomes obvious that many nodules, tender or non-tender which are frequently felt in fatty areas, and which were formerly referred to as fibrositic nodules, represent true fat herniations and thus constitute another but rare etiological agent producing symptoms which have been included by the author in the fibrositis syndrome.

LABORATORY FINDINGS

blood serum
eatinuria was
t being some-
what proportional to the symptoms.[3]

...tic patients, *Elliott*[13,14] *has shown*
le in some cases Robinson[10] has
ma volume. An elevation of blood
relationship to gout.[4]

In a case cured by *roentgen therapy*, no recurrence was noted for ten months following 8 treatments of 225 r each, at 25 cm. distance, 2 m.a intensity and using a filter of 6 mm Al. Treatments were given alternately to the internal and external surfaces of the affected knee. We have obtained temporary remission of the intermittent swelling with such radiotherapy. In the case quoted the remission lasted twelve months

Because of the analogy of periodic hydrarthrosis to local urticaria, ergotamine tartrate[35] has been advocated and favorable reports have followed its use parenterally and orally Since this is a chronic disease, *extreme caution* must be used if ergotamine is given over a period of weeks, and its use in such a syndrome does not seem warranted.

Histamine has not been of value in reported cases, the antihistaminic drugs such as pyribenzamine or benadryl have not been helpful in our experience. In one of our patients oral penicillin was given for two weeks without benefit

The use of intra-articular hydrocortisone acetate has been followed by somewhat variable results. In *several* instances, two injections of 25 mgm at the peak of swelling have led to a *complete cessation of the cycle with a remission lasting for four months* In one instance the same procedure was followed by a more frequent appearance of swelling *It would appear that this relatively innocuous procedure should be tried before roentgen therapy* (*See* Chapter 18 for details of technic)

BIBLIOGRAPHY

1 Baker, B M Arch Int Med , 44, 128, 1939
2 Benda, T Allg Med Centr Ztg , 69, 471, 483, 496, 508, 421, 1900
3 Berger, H Jour Am Med Assn , 112, 2402, 1939
4 Bierring, W L Jour Am Med Assn , 77, 785, 1921
5 Burchard, O Deutsch med Wchnschr , 28, 380, 1902
6. Cannone, N Quoted by ref 18
7 Collins, D H Jour Path and Bact , 42, 113, 1936.
8 Cook, C K Med Rec , 145, 300, 1937
9 Féré, D Rev neurol , 1, 465, 1893
10 Fridenberg, A H Med Rec , 33, 657, 1888
11 Ghormley, R K , and Deacon, A E Am Jour Roentgenol , 35, 740, 1936
12 Grandidier Berl klin Wchnschr , 9, 266, 1872
13. Hench, P S Proc Staff Meet Mayo Clin , 13, 161, 1938
14 Hench, P S , *et al* Ann Int Med , 8, 1685, 1935
15 ——— *Ibid* , 11, 1213, 1938
16 ——— *Ibid* , 28, 316, 1918
17 Klotz, P Gaz d hôp , 108, 1121, 1935
18 Krida, A Jour Bone and Joint Surg , 15, 449, 1933
19 Lewin, P., and Taub, S J Jour Am Med Assn , 106, 2144, 1936
20 Linberger, A Beitr z klin Chir , 30, 200, 1901
21 May, E , Huet, J A , and Barnaud, M Bull et mém Soc méd d hôp de Paris, 53, 106, 1937
22 Miller, J , and Lewin, P Jour Am Med Assn, 82, 1177, 1924
23 Moore, C H Lancet, 1, 485, 1864
24 Panas Bull et mém Soc de Chir de Paris, 4, 240, 1878
25 Paus, N Norsk Mag f Laegevidensk , 85, 760, 1924
26 Perrin, E. R Jour de méd , 3, 82, 1845
27 Porter, J L , and Lonergan, R C Jour Bone and Joint Surg , 14, 631, 1932
28 Queries and Minor Notes Jour Am Med Assn , 123, 249, 1943
28a Reimann, H A Medicine, 30, 219, 1951
29 Rowe, quoted by Bray, G W in Recent Advances in Allergy, Philadelphia, The Blakiston, Co , p 467, 1937.

Roentgen rays of the individual areas are negative Cultures of nodules or tissue removed at biopsy have shown no bacterial growth.

CLINICAL MANIFESTATIONS

Depending on the cause, the onset of fibrositis may be sudden or slow and insidious

Clinically, the syndrome is characterized by pain and stiffness, particularly on movement, occurring in the subcutaneous tissues, in fibrous origins, insertions and aponeuroses of muscles, in fascia, ligaments or tendons, in the fibrous portions of the joint capsule and in the supporting tissues of some nerves [15]

It may run an acute, subacute or chronic course, with remissions and exacerbations, the patient being relatively free for days at a time There is

muscles of the body because of pain consequent to the motion Pain on motion of the part and muscular stiffness are often the only symptoms Others may complain of sore, tender areas with occasional spasm of the surrounding muscles

The pain of fibrositis may be dull or sharp, continuous or intermittent.[21] It is occasionally knife-like or burning, like a hot poker The pain and stiffness are usually worse on awakening or after any period of inactivity for several hours, most symptoms are usually decreased following mild exercise The pain is usually a dull ache, most marked in the morning, it is usually associated with stiffness. The pain decreases and the stiffness lessens as the part is exercised The patient can, as it were, "work it off" but with the day's activity and the onset of fatigue it tends to recur toward evening. It frequently recurs throughout the day following any prolonged period of inactivity, such as sitting in a theater, sitting for long periods at a desk or table, etc [15] Some of these individuals are so stiff and sore after a full night's sleep that they deliberately are awakened by an alarm clock 2 or 3 times a night so that they can get up and walk around for a few minutes, to lessen their stiffness and soreness, after doing this, they feel less uncomfortable on awakening Special movements or pressure may cause sharp twinges of pain

In Ellman's 50 patients, pain was confined to a group of muscles in half of the cases Fourteen of these had accompanying pain in various parts of the body, and in another 6 there was also pain which travelled or

adjectives used included excruciating, agonizing, nagging and depressing [14]

The symptoms are often precipitated by cold, damp, drafts, sudden changes in the temperature and weather, or by unusual strain or exertion Emotional upsets seem to play a major part in initiating the attacks.

Relief is usually obtained at least temporarily by heat in various forms, mild activity, salicylates and alcohol.

30 SCHLESINGER, H Wien klin Wchnschr, 39, 68, 1926
[illegible] A Holder, Noth-
[illegible] dvances in Allergy,
[illegible]
34. SERVICE, W C Am Jour Surg, 37, 121, 1937.
35 SHARPE, J. C.. Ann Int Med, 9, 1431, 1936.
36. WEISMANN-NETTER, M R · Bull et mém Soc. méd. d hôp de Paris, 53, 909, 1929.
37 ———· *Ibid*, 53, 185, 1937

In time, general lassitude and easy fatigue form a prominent part of the clinical picture and other associated symptoms are sleeplessness, headaches and depression.

No constitutional disturbance is found such as fever, chills, loss in weight, etc. Physica

and stiffness are the

Palpation may dis

one finger, the so-called trigger point, or myalgic spot of fibrositis, considered by some to be the scars of past trauma or inflammation In some no abnormal tissue is felt, but in others tissue abnormalities may be palpated as rounded nodules, plaques, or elongated fibrous structures. The question then arises, "Are these areas responsible for the patient's symptoms?" Certain tests seem to indicate a direct relationship (1) Pressure on these areas may reproduce the patient's pain not only locally, but in the areas of his referred pain. (2) Injection of the area with local anesthetic may give complete relief, a full range of painless movement being restored (3) Pain may also be produced by increasing tension in the suspected str

people who do

no Copeman and Pugh[10] non-tender nodules were found with equal frequency in those with and those without fibrositis, but tender nodules or trigger points were found in only 3 per cent who did not give a history of fibrositis and in 30 per cent of those who did.

These nodules have probably been overstressed as a diagnostic aid; obvious visible nodules are rarely found. Krusen[17] questioned whether fibrositic nodules were as important as many writers believed, and whether they could be rubbed away as consistently as some stated. Buckley[4] also believes that the importance of fibrositic nodules has been overestimated, as they are often asymptomatic. Some have stated they are "only accessible to the finger of faith."[21]

Although some claim that they are able to palpate nodules in a large percentage of these patients, many physicians are able to detect nodules in only a very small percentage of such sufferers, even after the most careful

atrophy is also uncommon except where repeated attacks in the same area have resulted in disuse.

In many patients the symptoms are limited to certain well defined areas. The common *stiff neck* often associated with an occipital headache has been called **cervical fibrositis**. Many believe that pain in the shoulder girdle is often part of the fibrositic syndrome. In 105 of such cases, Kelly[24] found a supraspinatus trigger point in 62 and pressure on these points was said to reproduce the patient's pain not only locally but in referred areas down the arm and into the neck. In the lumbar area a common form of fibrositis is usually called **lumbago**, and *constitutes one of the common causes of low back pain.* In most cases tenderness is diffuse along the lower sacrospinalis muscles and in such cases, local injection has little value in the relief of

CHAPTER 35

THE FIBROSITIS SYNDROME
(NON-ARTICULAR RHEUMATISM)

By WALLACE GRAHAM, M.D

THE name fibrositis was first introduced by Sir William Gowers in 1904

denote the possible

he lumbar muscles.

was the underlying

pathology and he defined fibrositis as "a condition of chronic inflammation of the white fibrous tissue of the fascial aponeuroses, sheathes of muscles and nerves, ligaments, tendons, periosteum, and subcutaneous tissues, occurring in all parts of the body and giving rise to pain, aching, stiffness and other symptoms, the result of preceding general infections or local

findings which suggested the presence of chronic inflammation

For years in the United States the term fibrositis was received with reluctance and skepticism because of its ill-defined pathology Slocumb[33] first introduced the subject in America in 1936 and since that time it has emerged as the most controversial condition in the field of rheumatism. A review of the literature reveals an unusual number of authors vehemently defending a variety of theories in regard to its pathogenesis, but in all cases basic histological studies of the soft tissues, necessary in support of their thesis, are conspicuous by their absence.

It is unfortunate that the term fibrositis, which suggests an inflammatory etiology for a common rheumatic syndrome has crept into general use *No single cause has yet been determined and it is not established that inflammation is concerned in its pathogenesis* Lacking a firm pathological background the diagnosis of fibrositis has become a veritable depot for the problem cases of non-articular rheumatism, a situation which does not help to clarify the nature and identity of this very common complaint

It is probable that the syndrome represents a soft tissue reaction to a variety of different stimuli, traumatic, infectious, toxic, psychogenic, etc., and if this is the case the term fibrositis, used in its present broad sense, should be restricted in favor of an etiologic terminology which will lead to more fundamental treatment

CLASSIFICATION

Collins[5] has defined fibrositis as an acute, subacute, or chronic painful state of muscles, subcutaneous tissues, ligaments, tendons or fasciæ arising

pain. However, if a local tender area is found, dramatic relief is sometimes obtained by procaine injection

Gluteal fibrositis may be confused with the lumbar disc syndrome. In [illegible]

[illegible] logical signs of sciatic neuritis are absent

It should be stressed that although the prominent clinical finding in fibrositis is tenderness, it must be kept in mind that the patient's pain and tenderness may be referred and that the site of the local trigger area may be quite apart from the area in which pain is experienced. The actual position of the trigger area may be unknown to the patient and a careful search may be necessary to reveal this focal point.

Differential Diagnosis.—(*See also* Chapter on Psychogenic Factors in Rheumatic Diseases)

As already noted, the symptom complex of fibrositis may be initiated by a wide variety of disorders but it is important that the pain and stiffness of this group be distinguished from diseases which carry a much more unfavorable prognosis

The aching and pain of fibrositis may at first suggest early rheumatoid arthritis but the onset of intra-articular, extra-articular and systemic features characteristic of rheumatoid arthritis soon readily separate the two conditions

Because of the fear and anxiety which always accompany a diagnosis of arthritis, it should be stressed that this diagnosis should not be made until definite signs of a true joint lesion are manifest. Many forms of arthritis may have an associated peri-articular fibrositis which, of course, is related to the articular lesion Fibrositic pain in the chest wall will be readily distinguished from angina pectoris if an adequate history is taken. Epidemic Myalgia (devil's grip, epidemic pleurodynia, Bornholm disease) is an acute febrile illness whose major symptom is pleural and abdominal [illegible]

Painful Shoulder)

A febrile relapsing nodular non-suppurative panniculitis (Weber-Christian's Disease) has been described [1] Crops of subcutaneous nodules appear during episodes of fever, pathologic examination reveals that there is an associated peculiar type of fat atrophy The disease is most common in women Nodules are found most frequently on the thighs Death has been reported occasionally in this syndrome.

PROGNOSIS

This will naturally depend upon the causative factor, but in spite of the recurrent symptoms, patients are rarely incapacitated and the *reassurance that they are not suffering from a progressive articular lesion gives considerable comfort*

independently of gross anatomic disease from which pain might be *referred*. *The pain may be felt locally* at tender points or may be referred according to segmental *distribution*. He believes, along with many others, that fibrous tissue is not *the only* tissue concerned in the process of fibrositis, nor inflammation the only process involved

Classified according to location fibrositis has been separated on an anatomic basis into: (1) **Intramuscular**—also known as myositis or muscular rheumatism. (2) **Periarticular**—involvement of the joint capsule. (3) *Tendinous or fascial* [illegible] of the fibrous sheath of tendons. (4) [illegible] (5) **Perineural.** (6) **Panniculitis.**

[illegible] fibrositis has been considered as (1) **Primary**—without [illegible]

[illegible] gout, etc., the result of strain or trauma, exposure, occupation, postural and structural abnormalities, etc

However, in regard to *Primary* or *Idiopathic Fibrositis*, it is now felt by many that if the patient is adequately studied, not *only physically but* psychologically, a cause for the symptoms will not be lacking, and the use of the term *Primary Fibrositis* which only indicates inadequate investigation, will no longer be necessary.

INCIDENCE OF FIBROSITIS

Fibrositis is probably the most common form of acute and chronic rheumatism, but analysis of its incidence is rendered impossible by the elastic limits which define the syndrome in various countries The difficulty is well shown in the contrasting figures of the incidence of fibrositis in the British and American forces during World War II. Rheumatologists in America were startled in 1940 when Copeman called attention to the high incidence of fibrositis in the British forces. Other British reports have been correspondingly high.

Incidence in British Forces—Copeman, 1940, 70 per cent of rheumatic cases admitted to a British Army Hospital

Hutchison, 1942, 69 per cent of rheumatic cases in a British Army series

Savage, 1942, 52 per cent of rheumatic cases seen, 20 per cent of those admitted to a British Military Hospital.

Schmitt, 1942, 42 per cent of rheumatic cases seen in a study of miners.

Bach, 1947, 71 per cent of 100 cases seen as out patients at a British Rheumatism Clinic.

Incidence in American Forces—Boland and Corr, 1943, 5.8 per cent of 450 admissions to a U.S. Army Hospital.

Hollander, 1945, 3 per cent of 5,200 cases admitted to a U. S. Army Arthritis Hospital (see Chapter 1, p 23).

These studies of the relative frequency of rheumatic diseases among British troops indicate an incidence of fibrositis many times that seen in [illegible] -ble to [illegible] recog- [illegible] dis-crepancy in incidence is due to the fact that *no* cases of psychogenic rheu-

TREATMENT

First in importance is the education of the patient in regard to the nature of the lesion from which he is suffering. It is already obvious that the symptom complex of fibrositis may arise from numerous sources and the treatment must naturally be directed against the initiating agent whether it be systemic disease, some form of arthritis, the result of trauma, exposure or occupation, herniated fat or commonly a psychological fault

Most patients who have fibrositis think that they have arthritis and are filled with the fear and anxiety which accompanies this diagnosis **Reassurance** that the trouble does not represent a progressive, destructive joint disease lifts a great weight from the patient's mind, and is the most potent factor in treatment.

When a psychological fault has definitely been established as the cause, psychotherapy and not physiotherapy, in its ordinary sense, is indicated As Weiss[38] suggests "when we say to these people that their aches and pains and fatigue are due to the fact that they are always in a state of tension, that they do not know how to relax, even at night, and that because they are taut, their muscles are crying out in protest with aches and pains, it carries conviction and provides a stepping stone for them to begin to talk about their emotional problems." Such patients can often be shown from their own history that their symptoms increase when they are emotionally upset and subside as they gain mental and physical relaxation. They should be encouraged to carry on in spite of their symptoms and to try [illegible] and environmental

[illegible]

as a cure for their disorder but as a lesson in mental and physical relaxation.

After conference with the physical therapist, a "relaxing" program should be initiated The patient may soon learn that his symptoms disappear as his tautness and tension subside.

In other types of fibrositis, rest, heat and analgesics are often effective and the process of recovery may be hastened by massage. Exercises should be encouraged to increase circulation Krusen has for years advocated methods of home physiotherapy which are helpful The use of hot water, electric pad, and the infrared bulb are beneficial. Such measures increase the local blood supply and soothe the pain but must be followed by active exercises as soon as possible. Passive physiotherapy without action is disappointing.

Salicylates are very helpful but in many cases are inadequate Where trigger points are found, which on pressure reproduce the patient's symptoms, injection of a local anesthetic will occasionally give dramatic relief. Such injections occasionally relieve the local muscle spasm, and in relieving the pain temporarily movement is promoted and the symptoms may not return.

Infrared radiation can readily be applied at home, a simple type being [illegible] stance of eighteen inches [illegible] The application of heat should be followed by massage and [illegible] es.

Fibrositic patients should be protected against an undue amount of chilling, dampness, and sudden changes in temperature. Fatigue and exhaustion aggravate the symptoms and the patient's life must therefore be sensibly regulated

DISCUSSION

The introduction of the term fibrositis has fulfilled a useful purpose in separating the articular from the non-articular forms of rheumatism but confusion has arisen in the field of non-articular rheumatism due to the attempt to include a variety of widely separate disorders under the common term fibrositis, because such disorders give rise to a similar symptom complex

As Himsworth[23] has recently pointed out, the same syndrome may arise from different causes because interference in a chain of physiological processes at any point may produce the same impairment of bodily function. This thesis would appear to be fully applicable to the field of non-articular rheumatism.

The symptom complex of fibrositis which clinically is characterized by pain, stiffness and tenderness in the fibrous tissues without constitutional disturbance and without an established pathological picture, may be produced by a variety of etiologic agents already outlined

If effective treatment follows on accurate diagnosis, which is surely the case, the various components of non-articular rheumatism should be classified according to etiology and not thrust into the grab-bag labelled fibrositis

It should seem reasonable that the term fibrositis should be confined to those cases in which an inflammatory basis can be established, and if it is

physical and psychological investigation which his problem demands and this will lead to a more positive approach to treatment

BIBLIOGRAPHY

1 Bailey, R J Jour Am Med Assn, 109, 1419, 1937
2 Boland, E W, and Corr, W P Jour Am Med Assn, 123, 805, 1943
3 Brøchner-Mortensen, K, and Clemmensen, S Acta med Scandinav, 106, 540, 1941
4 Buckley, C W Ann Rheum Dis, 2, 83, 1940
5 Collins, D H Ann Rheum Dis, 2, 114, 1940
6 Copeman, W S C Brit Med Jour, 2, 263, 1943
7 ——— Brit Med Jour, 2, 191, 1949
8 C t Jour Med, 13, 37, 1944
9 —
10 C 2, 553, 1945
11 C
12 [illegible]
13 E
14 ——— Ann Rheum Dis, 4, 22, 1944
15 Ellman, P, et al Ann Rheum Dis, 3, 56, 1942
16 Ellman, P, and Shaw, D Ann Rheum Dis, 9, 341, 1950
17 Freyberg, R H Clinics, 2, 1586, 1944

of six to twelve days between the administration of serum and the onset

to distinguish from the trismus due to tetanus

Treatment.—Since the illness is self-limited, treatment need only be symptomatic. This, however, is important, because of the very distressing nature of the symptoms For the pruritic and urticarial features 0.3 to 0.6 cc of a 1:1000 solution of epinephrine should be tried, and, if effective, then the long-acting solutions of epinephrine in gelatin or oil may be continued More recently, the antihistaminics such as benadryl and pyribenzamine in doses of 50 to 100 mgms every six to eight hours have proved very effective in most cases, and when so, are recommended regularly for the expected duration of the disease

these new agents only in those cases in which the symptoms are so severe *as to be uncontrolled by antihistaminics and epinephrin*

The older treatments with calcium lactate, histaminase, ephedrine and soothing applications such as calamine lotion with phenol are relatively ineffective.

PERIARTERITIS NODOSA

This condition, first described by Kussmaul and Maier[15] in 1866, is a member of the collagen group of diseases and is of importance to rheumatologists because one of its most constant clinical features is joint and muscle pain Polyarteritis nodosa and necrotizing vasculitis are alternate names which are more apt from the point of view of the pathology but which have not so far come into general use Periarteritis nodosa is a rare disease with a total of only about 400 proved cases reported to date It occurs predominantly in adult males and is most common in the third and fourth decades

Pathology

may be fatal, all occur

The adventitia and loose perivascular tissues are infiltrated with inflammatory cells giving the picture of periarteritis for which the disease was

18 Gordon, R G.: Brit. Med. Jour., 2, 1083, 1935
19 ———. Ann. Rheum Dis., 2, 89, 1940
20 Halliday, J L.: Practitioner, 152, 6, 1944.
21 Hench, P. S, *et al* Ann Int Med, 10, 754, 1936
22 ———: *Ibid*, 28, 66, 1948
23. Himsworth, T. C · Quoted by Copeman.[7]
24 Kelly, M.· Med Jour. Australia, 1, 488, 1942.
25 ——— Ann. Rheum. Dis, 4, 46, 1944.
26. ———· *Ibid*, 5, 1, 1945
27. Krusen, F H.. Arch. Phys Ther., 18, 687, 1937.
28 Patterson, J. W. T.· Quoted by Douthwaite[12]
29 Race, J · Ann. Rheum. Dis, 2, 127, 1940
30 Robinson, G. L.: Ann Rheum Dis, 3, 207, 1943.
31 Savage, O · Brit. Med Jour., 2, 336, 1942
32. Schmidt, L Brit Jour Phys Med, 5, 145, 1942
33 Slocumb, C H · Proc. Staff Meet, Mayo Clinic, 11, 363, 1936, Jour Lab. and Clin Med, 22, 56, 1936
34. Steinberg, C. L Jour. Bone and Joint Surg, 24, 411, 1942
35 Stockman, R.. Ann. Rheum Dis, 2, 77, 1940.
36 Tegner, W Practitioner, 157, 446, 1946
37. Walters, J. A, Slade, H C, and Graham, W: Rheumatic Diseases, Philadelphia, W B Saunders, 1952
38. Weiss, E. *Rheumatic Diseases, Philadelphia*, W. B. Saunders, 1952.

in varying degrees. In some instances they may even be indistinguishable from it.[30]

The lesions may occur in the smaller arteries anywhere in the body and may be few in number or countless. Ischemia, mild or severe may occur in any organ through involvement of its attending artery so that an infinite variety of clinical manifestations may result. The kidneys, coronary arteries, mesenteric arteries and vessels of the extremities are commonly involved. The brain, lung, liver and spleen are almost as frequently involved

Pathogenesis.—As with the other collagen diseases, the pathogenesis and etiology of periarteritis nodosa is unknown. Before the Wassermann reaction permitted accurate diagnosis, syphilis was long suspected, and, later on, streptococcal and viral infections were proposed as the cause, but have never been proved Since Rich[20,21,22] reported the lesions of periarteritis in patients who died of infectious diseases but in whose cases he demonstrated ante-mortem sensitivity to serum, sulfonamides and iodine, the general trend of opinion has favored some sort of allergy or hypersensitivity rather than infection. The similarity of these lesions to those produced by injections of heterologous serum in animals has already been noted, also their resemblance to some of the lesions of serum sickness in humans. Why certain patients should respond in this manner to stimuli which in most persons are harmless is, of course, the crux of the problem.

The Clinical Picture.—The onset may be sudden or insidious. The course is usuall ... fatal termination ... cases it is short ... The general ... e infec- ... , sweating, weight loss and joint and muscle pains. The patient may present a baffling problem in diagnosis until the signs and symptoms of regional or local damage to organs due to arterial occlusion appear.

As would be expected from a knowledge of the pathology of this disease, the clinical manifestations may be exceedingly varied depending on the number, site and degree of the arterial occlusions. Infarctions in the myocardium give their usual signs and symptoms and may lead to heart failure, infarctions in the kidney may cause local pain, hematuria and hypertension, and occlusions of the cerebral vessels may cause hemiplegia, headache and a variety of cerebral symptoms depending on their site and size. Occlusion of the mesenteric arteries may cause abdominal pain, diarrhea, bleeding and gangrene of the bowel. Infarcts in the spleen, liver and pancreas are not uncommon and are associated respectively with pain and jaundice. Pain in the extremities is common and may be on the basis of vascular occlusion. But in 52 per cent of cases studied by Lovshin[17] ... and frequently early mani- ... fibers may be affected so ... in the lower extremities should direct our attention to the possibility of periarteritis nodosa.

Abdominal distress, albuminuria, hypertension, cardiac hypertrophy and arthralgia are almost always present at some stage of the disease.

PART V

THE DISEASES OF COLLAGEN

CHAPTER 36

THE COLLAGEN DISEASES

By John Lansbury, M.D.

Yardumian and Klemerman[30] have recently stated that the vascular lesions of disseminated lupus, rheumatoid arthritis, periarteritis nodosa, thromboangiitis obliterans and scleroderma are histologically identical Sub-

is that they are basically diseases of the connective tissue system, a system which can react to damage in only a limited number of modes, one of which is fibrinoid change *Collagen, as referred to here, denotes all inter-*

erythematosus, rheumatic fever, rheumatoid arthritis and its variants,

this

by

some, but will not be considered here since they have no rheumatic clinical

As the symptoms due to one episode of arterial occlusion subside, new symptoms in an unrelated part of the body may begin This migratory pattern of events may be of considerable aid in diagnosis Edema of the legs is common Absent pulsations in the vessels of an arm or leg, palpable nodulations of superficial arteries and indurations in the muscles or skin are helpful physical signs

Features Which Aid in Distinguishing Periarteritis Nodosa from Other Members of the Collagen Group of Diseases

1 About 70 per cent of cases occur in adult males
2 The disease may be precipitated by a variety of drugs such as sulfonamides, etc
3 Neuritic leg pains are present in about 50 per cent of cases
4 Kaleidoscopic clinical picture due to arterial occlusions in unrelated parts of the body
5 Hypertension secondary to renal disease and possibly to multiple arteriolar occlusions
6 Palpable tender nodules in muscles and occasionally arteries
7 Biopsy diagnostic when positive
8 Eosinophilia in some cases

Laboratory Findings.—The urinary findings resemble those of nephritis

and Cooper[5] have described a peculiar change in the roentgenogram of the

clinical as well as pathological features in common. Indeed in the early stages it may be exceedingly difficult to make a clinical distinction between them, also a careful study not infrequently reveals features of several members of the group co-existing and overlapping in the same patient.

When the diagnosis is not clear, it is unwise to force the diagnostic issue and the term "*Undifferentiated Collagen Disease*" is a useful label With the possible exception of calcinosis, the onset of these diseases *may* follow some damaging stimulus such as infection, injection of foreign protein, exposure to sunlight and other physical agents, drugs[9] and emotional stress. [illegible]

because of the short duration of the disease.) Joint pain and swelling are present in all these diseases and are the presenting symptom in the majority of them. The scleroderma group is possibly excepted since the joint disabilities are due mainly to contractures of the attending structures, although in some instances rheumatoid arthritis appears to co-exist. Renal, cardiovascular and pulmonary lesions are also frequently encountered. The course of these diseases tends to be subacute or chronic with remissions which are especially striking in rheumatic fever and lupus In the majority of these diseases the serum gamma globulin is increased, and serum albumin is decreased. The active manifestations of most of these diseases is suppressed by corticotropin, cortisone and hydrocortisone.

Features Which are Common to the Majority of the "Collagen" Diseases

(Dermatomyositis, Periarteritis Nodosa, Disseminated Lupus, Rheumatic Fever, Rheumatoid Arthritis, Scleroderma)

1. Chronic, relapsing, generally life long course.
2. Onset and acute exacerbations often precipitated by stress of various kinds.
3. Clinical features suggesting an acute, sub-acute or chronic infection, but no causal organism demonstrable.
4. Active phases of the diseases associated with fever, anorexia, weight loss, weakness.
5. Arthritis, vascular disturbances, cardiac, pulmonary, muscular and renal lesions of varying prominence
6. Rheumatic nodules, lymphadenopathy
7. Fibrinoid change in collagen
8. Increased sedimentation rate.
9. Increase in globulins (especially gamma globulin) and decrease in serum albumin.
10. Active features of the disease suppressed by corticotropin, cortisone and hydrocortisone, but the natural course of disease not arrested and may even be accelerated in certain instances

with the "primary" rheumatic diseases. The serial involvement of such widely separated organs as the brain, heart, intestines and kidneys should heighten one's suspicion that periarteritis is present. The detection of palpable pulsating nodules on the superficial arteries and a positive biopsy are pathognomonic, but such nodes are often absent, and random biopsies negative.

Treatment.—There is no recognized curative treatment. Goldman[9] reported an apparent cure following sulfapyridine, and Goodman[10] reported one case in which the disease followed the administration of sulfadiazine but which was apparently cured by a subsequent dose of the same drug. Treatment with sulfonamides would seem highly illogical, since they were found to be the *precipitating* agent in several of Rich's cases, and since a history of previous medication of this type is often obtained from these patients.

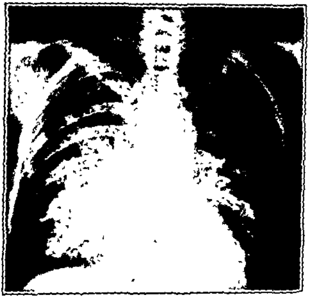


FIG. 193.—Chest film of a patient having respiratory symptoms with periarteritis nodosa. Note massive and fairly symmetrical mottled density extending out from the hilum to middle lung zone—the so-called corona. (Barden and Cooper, courtesy Radiology.)

The active phase of periarteritis nodosa can be reversed by ACTH and cortisone, and these agents seem to be the only ones which have any constant effect on the disease. However, the initial benefit is often deceptive since the course of the disease seems to be unaffected. If a "cure" results it would seem to be because the patient has been protected from the lethal features of his disease long enough for spontaneous recovery to take place.

Unfortunately, even when healing of the arterial lesions takes place under cortisone therapy the end result may be additional degrees of vascular occlusion which cause widespread infarctions. Examples of this effect were reported by Baggenstoss *et al.*[2] Progressive hypertension is another complication of cortisone therapy in this disease.

Clinical Dissimilarities.—The clinical picture of these diseases is well-correlated with the pathological findings, which, although characterized by the common denominator of collagen degeneration, are distinct in that the lesions vary in over-all type and location, with, however, considerable overlapping in some cases. Clinically, the emphasis falls on the joints in rheumatoid arthritis, on the heart in rheumatic fever, on the skin, enteric canal and lungs in scleroderma and on the fascial planes of the musculature in dermatomyositis. Calcium retention is a feature of the last two, also in calcinosis. In disseminated lupus the emphasis is on the serous membranes and vasculature, and, in periarteritis nodosa the whole vascular

Thus, *the whole skeletal and connective tissue system of the body is embryo-*

produces gelatin. It exists in large amounts in bone, cartilage, corium, and *the intercellular substance of connective tissues in general.* Apparently, it is a complex structure of protein linked with a mucopolysaccharide, such as hyaluronic acid. In cartilage the chief mucopolysaccharide is chondroitin-sulfuric acid. *Hyaluronic acid* is found in the interfibrillar substance of connective tissue, in many of the tissue clefts, particularly the joint spaces, vitreous of the eye, umbilical cord, etc. This mucopolysaccharide is composed of glucosamine and glucuronic acid.

There is a highly complicated enzyme system regulating the hyaluronate containing mucopolysaccharides in which various hyaluronidases are

article which is well worth consulting.[6]

Pathology of Collagen.—The fundamental morphological alteration in

change.[14] In addition, the fibroblasts undergo varying degrees of proliferation, degeneration and necrosis. Fibrinoid change is found at the center

It is impossible to state whether ACTH and cortisone are to be recommended here In the face of a progressively downhill course and in the absence of renal involvement they are probably indicated in palliative doses

DISSEMINATED LUPUS ERYTHEMATOSUS

Disseminated lupus erythematosus was described by Kaposi in 1872,[12] and was first regarded as a skin disease of tuberculous etiology and as a variant of chronic discoid lupus Subsequent studies[4,13,15] have revealed it to be a relatively common, highly important and highly lethal generalized connective tissue disease which has most of the features common to the collagen necrosis group of diseases Since the recent increased interest in the rheumatic diseases, and particularly since the discovery of the L E cell by Hargraves *et al.*[11] many cases of the subacute and chronic forms of the disease which would previously have been diagnosed as rheumatic fever or rheumatoid arthritis have been identified as disseminated lupus erythematosus

The name, Disseminated Lupus Erythematosus, is unfortunate since it implies a tuberculous etiology which has never been proved The dermatologists have proposed shortening the name to "Erythematodes" which to us seems even less apt A name such as "Remittent Serovasculitis," which is reasonably descriptive, both from a clinical and pathological viewpoint, and which distinguishes it from other collagen diseases, would seem preferable.

Etiology.—The disease has a marked predilection for young females The ratio is estimated as 95 per cent females to 5 per cent males affected It is most common in the second and third decade of life seldom occurring before the age of fifteen About 90 per cent of recognized cases die within five years The cause is unknown, and, as in the other rheumatic diseases, no infective agent has ever been proved despite clinical features, such as fever and increased sedimentation rate, which suggest infection The disease is not transferable to animals Attempted treatment by castration and testosterone have failed to implicate the ovary as an endocrine cause, although the predilection for females cannot be denied The acute phase of the disease may be precipitated by various types of stress Among these, the specifically harmful effect of the exposure to sunlight is important both from the point of view of pathogenesis and clinical management

Pathology.— [illegible]

from Aschoff bodies are not infrequently found, and the vasculature of the kidneys, spleen and skin show necrosis of the connective tissue matrix of the vessel walls with endothelial proliferation and occlusion Enlargement and focal necrosis of the lymph nodes occurs in about 50 per cent of cases

There have been few studies of the pathology of the affected joints Hypertrophy of the synovial villi has been reported with perivascular infiltration of inflammatory cells of the subsynovial and capsular tissues

in lupus erythematosus, scleroderma and dermatomyositis. Fibrinoid degeneration also occurs in diseases with an obvious "allergic" pathogenesis such as serum sickness and drug sensitivity.

Pathogenesis of the Collagen Diseases.—The connective tissue system is composed of hyaluronate containing ground substance in the form of a gel, fibroblasts which are believed to be the source of the former, and various fibers and matrices which seem to be condensations and modifications of the ground substance

The collagen diseases are manifested by widespread changes in this connective tissue system. However the similarities which exist between these changes may indicate a limited and stereotyped reaction to damage rather [illegible] sma cells [illegible] iem as a [illegible]

However, the actual etiology behind this remains unknown, and any attempt to identify it must of necessity be purely speculative. Steroid hormones, such as estrins, androgens and corticosteroids are known to regulate the activity of fibroblasts and hyaluronate containing tissues in various ways; thyrotropic and growth hormones also affect the connective tissue system. Yet, at present, no disturbance in those hormonal regulators of the connective tissues has been proved to be the cause of the collagen diseases

Among the vitamins, ascorbic acid is known to be necessary for the functioning of fibroblasts, yet subclinical scurvy is not a feature of the rheumatic deseases, neither are they cured by the administration of Vitamin C. In rheumatic fever, upper respiratory tract infections by group A hemolytic streptococci are unquestionably a precipitating factor, but other, as yet unknown factors, are necessary for the development of the disease. The case for infection in general in these diseases has narrowed down to the possibility of some hypothetical virus or pleuro pneumonia-like organism, or to some disturbance in the immunity mechanism which causes the body to over-react to infections which would be otherwise "normal" or harmless.

[illegible]

planation" we must remember the sober fact that the true nature of allergy remains a mystery, and that we are in danger of attempting to explain one unknown in terms of another Ordinary clinical allergy is not especially associated with the Collagen Diseases.

Recently an association between neoplasia and rheumatism has been noted. Polley[19] has reported the occurrence of joint symptoms and/or signs in 18 of 21 cases of localized fibro-mesothelioma of the pleura. Joint [illegible] l arthritis have [illegible] of neoplasms,[15a] and acute dermatomyositis is likewise by no means rare as a complication of malignancy.[4a] Disseminated lupus has also been found in the same situation This observation, although of great interest only adds to the mystery of the pathogenesis of the collagen diseases. It would seem that

with th- "--'- ---" -' - - -'- !' - ... .
widely
heighte
palpable pulsating nodules on the superficial arteries and a positive biopsy are pathognomonic, but such nodes are often absent, and random biopsies negative.

Treatment.—There is no recognized curative treatment. Goldman[9] reported an apparent cure following sulfapyridine, and Goodman[10] reported one case in which the disease followed the administration of sulfadiazine *but which was apparently cured by a subsequent dose of the same drug.* Treatment with sulfonamides would seem highly illogical, since they were found to be the *precipitating* agent in several of Rich's cases, and since a history of previous medication of this type is often obtained from these patients.

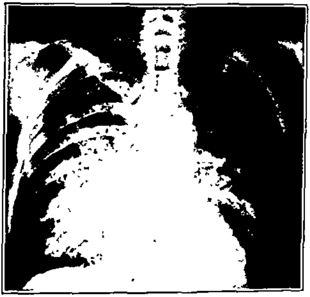


FIG 199 —Chest film of a patient having respiratory symptoms with periarteritis nodosa Note massive and fairly symmetrical mottled density extending out from the hilum to middle lung zone—the so-called corona (Barden and Cooper, courtesy Radiology)

The active phase of periarteritis nodosa can be reversed by ACTH and ... which have any con- ... n deceptive ... ure" results it would seem to be because the patient has been protected from the lethal features of his disease long enough for spontaneous recovery to take place.

Unfortunately, even when healing of the arterial lesions takes place under cortisone therapy the end result may be additional degrees of vas- ... widespread infarctions. Examples of this effect ... ssive hypertension is another ... ease.

clarification of the problem must await further studies of the normal and disturbed physiology of the connective tissue system.

Features of Rheumatoid Arthritis Which Tend to Distinguish it from Other Members of the Collagen Group of Diseases

1 Prodromes of exhaustion, fatigue and psychological stress elicited in about 80 per cent of cases
2 The articular manifestations usually overshadow the systemic manifestations
3 Inflammatory ocular lesions (iritis, uveitis, etc) common in rheumatoid arthritis, rare in other collagen diseases
4 Blood pressure is normal or low in most cases
5 Serum contains an unidentified factor which agglutinates various bacteria, collodion particles and tissue extracts, but does not contain antibodies against type A streptococcal products as in the case of rheumatic fever
6 Amyloidosis occurs in about 10 per cent of later cases
7 Characteristic nodules at pressure points (*e g* , elbows)

SERUM SICKNESS

Serum sickness is a man-made disease which dates from the introduction of diphtheria anti-toxin in 1890. It was first thought to be due to the antitoxin itself but was shortly shown to be caused by the serum since normal horse serum produces the symptoms with equal ease As immune sera have become more refined, serum reactions and serum sickness have become less frequent The anaphylactic shocks which may be fatal and

susceptible then the colored races but there are no preferences for sex or age

Pathogenesis.—There can be no doubt as to the 'allergic' etiology of serum sickness and no question of infection as its cause. Yet both the pathology and the clinical manifestations of this syndrome follow the general pattern of the rheumatic and collagen diseases. This seems very strong evidence as to the probable role of altered immunity in this whole group of diseases.

Although, in general, the incidence of serum sickness depends on the amount of serum given, the production of symptoms in a particular case parallels the titer of precipitins which are developed during the so-called incubation period. Thus, the manifestations of the disease seem to be dependent on the amount of immunity rather than on the amount of antigen. The significance of this is not altered by the fact that, generally, large amounts of antigen produce correspondingly large amounts of antibody.

The Clinical Picture.—The symptoms of serum sickness begin from one to two weeks after the administration of the serum. Rarely, they are

It is impossible to state whether ACTH and cortisone are to be recommended here. In the face of a progressively downhill course and in the absence of renal involvement they are probably indicated in palliative doses.

DISSEMINATED LUPUS ERYTHEMATOSUS

Disseminated lupus erythematosus was described by Kaposi in 1872,[12] and was first regarded as a skin disease of tuberculous etiology and as a variant of chronic discoid lupus Subsequent studies[4 13 14] have revealed it to be a relatively common, highly important and highly lethal generalized connective tissue disease which has most of the features common to the collagen necrosis group of diseases. Since the recent increased interest in the rheumatic diseases, and particularly since the discovery of the L E cell by Hargraves *et al* [11] many cases of the subacute and chronic forms of the disease which would previously have been diagnosed as rheumatic fever or rheumatoid arthritis have been identified as disseminated lupus erythematosus

The name, Disseminated Lupus Erythematosus, is unfortunate since it implies a tuberculous etiology which has never been proved The dermatologists have proposed shortening the name to "Erythematodes" which to us seems even less apt A name such as "Remittent Serovasculitis," which is reasonably descriptive, both from a clinical and pathological viewpoint, and which distinguishes it from other collagen diseases, would seem preferable

Etiology.—The disease has a marked predilection for young females The ratio is estimated as 95 per cent females to 5 per cent males affected It is most common in the second and third decade of life seldom occurring before the age of fifteen About 90 per cent of recognized cases die within five years The cause is unknown, and, as in the other rheumatic diseases, no infective agent has ever been proved despite clinical features, such as fever and increased sedimentation rate, which suggest infection The disease is not transferable to animals. Attempted treatment by castration and testosterone have failed to implicate the ovary as an endocrine cause, although the predilection for females cannot be denied The acute phase of the disease may be precipitated by various types of stress Among these, the specifically harmful effect of the exposure to sunlight is important both from the point of view of pathogenesis and clinical management

Pathology.—Despite the devastating clinical picture, few gross lesions are found at necropsy. The actual lesions are disseminated in the collagenous tissues underlying the serous surfaces of the endocardium, pericardium, joints, pleura and peritoneum. Focal myocardial lesions which are distinct from Aschoff bodies are not infrequently found, and the vasculature of the kidneys, spleen and skin show necrosis of the connective tissue matrix of the vessel walls with endothelial proliferation and occlusion Enlargement and focal necrosis of the lymph nodes occurs in about 50 per cent of cases.

There have been few studies of the pathology of the affected joints. Hypertrophy of the synovial villi has been reported with perivascular infiltration of inflammatory cells of the subsynovial and capsular tissues

delayed until the third week. They may appear early in patients who have been previously sensitized. The illness usually runs a course of seven to ten days but may persist for three weeks. Relapses may occur which are explained on the basis of antibodies developing at different rates for different antigenic fractions of the serum.

Generalized mild lymphadenopathy with variable tenderness is one of

of a subcutaneous injection of serum. Diffuse or macular erythemas may also occur, especially in relapses, and *purpura* is occasionally found in severe cases. These cutaneous manifestations are usually of short duration.

Edema of the face and extremities resembling that of acute glomerulonephritis is accompanied by signs of renal damage such as oliguria, albumin and casts in the urine and diminished renal function and occurs to some degree in about one-third of cases. Fever is also present in about a third of cases and roughly parallels their severity There is nothing characteristic about the temperature curve. It may be low or as high as 104° F.

Joint pain and stiffness are especially apt to occur after large amounts of serum and in severe cases. It may be the outstanding feature of the illness from the patient's point of view and may be the last complaint to disappear The large and medium joints are most commonly affected. Effusions occur in severe cases and may be multiple with a strong resemblance to rheumatic fever The joint fluid may contain as many as 20,000 leukocytes per cu. mm in which polymorphs predominate Serologic examination of the fluid may detect the presence of horse serum.

The illness ends by lysis and leaves no sequels except a sensitivity to

normal

Features of Acute Rheumatic Fever Which Aid in Distinguishing it from Other Members of the Collagen Group of Diseases
1. In 90 per cent of cases the first attack occurs under 15 years of age.
2. Acute episodes commonly follow an upper respiratory infection with ... interval of 0 to 14 days
3
4
5
6 ... ses
7 ... al)
8 Apparently complete remissions for months or years
9. High serum titres of antifibrinolysin, antihemolysin, antistreptolysin, antistreptohyaluronidase

As in rheumatoid arthritis the joint fluid is of low viscosity, suggesting depolymerization of the normal mucopolysaccharides.

Features of Disseminated Lupus Erythematosus Which aid in Distinguishing it from Other Members of the Collagen Group of Diseases

1. 95 per cent of cases occur in young females.
2. Acute phase may be precipitated by exposure to sunlight.
3. Effusions from any serous surface are common.
4. High fever and great "toxicity" out of proportion to joint findings.
5. Cerebral manifestations—unreasonable anxiety, convulsions
6. [illegible]
7. [illegible]
8. [illegible]
9. [illegible]
10. [illegible]

globulins and *presence of "L.E." cells*

Symptoms and Course.—Although the manifestations of this disease are very variable, three phases with reference to the joint manifestations can be distinguished as follows:—(1) An acute, fulminating form in which systemic features predominate and in which joint findings are minimal or absent. This may occur *de novo* or may occur as an acute exacerbation of the subacute or chronic phase. (2) A subacute form, which is clinically difficult to distinguish from early, active rheumatoid arthritis and rheumatic fever. In these cases the systemic manifestations, though less marked, are out of proportion to the joint findings. (3) A chronic form, in which the joint findings [illegible] toid arthritis, and in which [illegible] nly by special attention to [illegible] of L.E. cells, if present.

Actually not as much is known as would be expected about the course of this disease It is probably a lifelong affliction which is subject to spontaneous remissions with, however, the constant threat of acute exacerbations. In general, the acute and subacute forms are most rapidly fatal but less is known about the course of the chronic forms. There is evidence of some smouldering activity even during an apparent remission The onset may follow exposure to sunlight and is characterized by malaise, increasing fatigue, weight loss and fever. In some cases, the history begins with pulmonary manifestations mistaken for virus pneumonia Arthralgia and a slowly shifting polyarthritis with joint and muscle pain is common and may resemble the arthritis of rheumatic fever, but responds poorly to salicylates. In the course of the disease, pleural, pericardial and peritoneal effusions with local pain and tenderness may come and go. As the severity of the disease increases, the fever rises to 103° to 105° F. with a diurnal variation of 1° or 2°. In some cases the peak of fever may occur

Diagnosis.—Rheumatic fever, the onset of any type of polyarthritis and the collagen diseases in general, as well as glomerulonephritis have to be considered in the differential diagnosis. However, the presence of itching and urticaria, together with the short course of the disease and the interval of six to twelve days between the administration of serum and the onset of symptoms render the diagnosis easy in most cases. Relapses or complications of the original infection for which the serum was given should be kept in mind. Involvement of the temporo-mandibular joint may be difficult to distinguish from the trismus due to tetanus.

Treatment.—Since the illness is self-limited, treatment need only be symptomatic. This, however, is important, because of the very distressing nature of the symptoms. For the pruritic and urticarial features 0.3 to 0.6 cc. of a 1:1000 solution of epinephrine should be tried, and, if effective, then the long-acting solutions of epinephrine in gelatin or oil may be continued. More recently, the antihistaminics such as benadryl and pyri- [illegible] oved [illegible] for

As would be expected, ACTH and cortisone suppress the manifestations of serum sickness.[4] Theoretically, this being a short term, self-limiting disease, it should be an ideal situation in which to take advantage of the suppressive action of ACTH and cortisone. However, we recommend these new agents only in those cases in which the symptoms are so severe as to be uncontrolled by antihistaminics and epinephrin.

The older treatments with calcium lactate, histaminase, ephedrine and soothing applications such as calamine lotion with phenol are relatively ineffective.

PERIARTERITIS NODOSA

This condition, first described by Kussmaul and Maier[15] in 1866, is a member of the collagen group of diseases and is of importance to rheumatologists because one of its most constant clinical features is joint and muscle pain. Polyarteritis nodosa and necrotizing vasculitis are alternate names which are more apt from the point of view of the pathology but which have not so far come into general use. Periarteritis nodosa is a rare disease with a total of only about 400 proved cases reported to date. It occurs predominantly in adult males and is most common in the third and fourth decades.

Pathology.—The fundamental lesion [illegible]

may be fatal, all occur.

The adventitia and loose perivascular tissues are infiltrated with inflammatory cells giving the picture of periarteritis for which the disease was originally named. The general vascular lesions of serum sickness, rheumatic fever, disseminated lupus erythematosus, thromboangiitis obliterans and the renal lesions of scleroderma resemble those of periarteritis nodosa.

in the morning rather than the afternoon. Apprehension may be out of proportion to the other symptoms, and towards the end the patient is highly "toxic" with various combinations of irritability, disorientation muscle twitchings and tremors, stupor, amblyopia and convulsions, al- [illegible] Death occurs from

generally, but not always, occur before the skin lesions. There are transient, migratory swellings with some heat involving several joints often with remissions. There is some tenderness, stiffness and limitation of



motion *In some cases the joint manifestations are the outstanding feature* of the disease and, in subacute and chronic cases they may persist for months or even years Late in the disease permanent joint changes causing deformity especially in the small joints are occasionally seen.[8, 29] In such cases the co-existence of rheumatoid arthritis is difficult to disprove. Slocumb[28] notes the occurrence of arthritis in 60 per cent of acute and subacute disseminated lupus and in 20 per cent of the chronic cases

It will be seen from the above, that the arthritis of disseminated lupus has no very characteristic features so that one must rely on the general

delayed until the third week. They may appear early in patients who hav been previously sensitized. The illness usually runs a course of seven to te days but may persist for three weeks. Relapses may occur which ar explained on the basis of antibodies developing at different rates for different antigenic fractions of the serum.

Generalized mild lymphadenopathy with variable tenderness is one o [illegible]

of a subcutaneous injection of serum. Diffuse or macular erythemas may also occur, especially in relapses, and purpura is occasionally found in severe cases. These cutaneous manifestations are usually of short duration.

Edema of the face and extremities resembling that of acute glomerulonephritis is accompanied by signs of renal damage such as oliguria, albumin and casts in the urine and diminished renal function and occurs to some degree in about one-third of cases. Fever is also present in about a third of cases and roughly parallels their severity. There is nothing characteristic about the temperature curve. It may be low or as high as 104° F.

[illegible]

in severe cases and may be multiple with a strong resemblance to rheumatic fever. The joint fluid may contain as many as 20,000 leukocytes per cu mm in which polymorphs predominate. Serologic examination of the fluid may detect the presence of horse serum.

The illness ends by lysis and leaves no sequels except a sensitivity to [illegible]

tance. There is seldom a significant leukocytosis. An eosinophilia may occur but is an inconstant finding. The sedimentation rate is usually normal.

Features of Acute Rheumatic Fever Which Aid in Distinguishing it from Other Members of the Collagen Group of Diseases

1. In 90 per cent of cases the first attack occurs under 15 years of age.
2. Acute episodes commonly follow an upper respiratory infection with Group A hemolytic streptococci after an interval of 9 to 14 days.
3. Arthritis of the rapidly migrating pattern.
4. Unique response of fever to salicylates.
5. Past or present history of chorea.
6. Erythema marginatum—seldom if ever found in other collagen diseases.
7. Electrocardiographic evidence for myocarditis (prolonged P-R interval).
8. [illegible]
9. [illegible] antistreptolysin,

characteristics of the disease for the diagnosis. (Even in the acute phase of the disease roentgenograms of the joints may show rarefaction which is

ill-advised surgical exploration The spleen is infrequently palpable and lymphadenopathy is inconstant. The liver may at times be enlarged and



ight icterus may occur. In advanced cases, there may be moderate edema nd moderate arterial hypertension, and rarely, the ocular fundi show idespread vascular and perivascular changes with arteriolar segmentation, emorrhages, exudates and venous thrombosis *Although these changes* irror the general vascular damage disseminated throughout the body, ey are not of themselves pathognomonic of lupus.

At some time during the course of the disease, characteristic skin lesions sually appear although they may be absent throughout. Often, they

follow exposure to sunlight. They consist of a confluent, macular, red eruption on the exposed surfaces. A "butterfly" distribution over the bridge of the nose and malar eminences in which the forehead, chin and pinnae may share is characteristic, although other skin lesions may have a similar distribution (Figs. 200 and 201.) The fingers, a v-shaped area on the upper anterior chest and various pressure points may also be involved. These lesions may disappear completely with a remission or may leave pigmented areas. There may be shallow ulcerations on the buccal mucosa and in the severe terminal phases generalized swelling and oozing of blood from the oral cavity. This may be secondary to thrombocytopenia.



Fig. 202.—A typical group of L.E. cells (neutrophils distorted by a large amount of phagocytosed material) from a preparation of plasma from a patient with systemic lupus erythematosus, incubated with normal human bone marrow. Note nucleus of each "L.E." cell is forced to periphery by ingested material in cytoplasm. (About × 900.) Courtesy Drs. A. J. Creskoff and W. R. Bell.

Laboratory Findings.—Leukopenia averaging 3500 WBC per cubic mm., and sometimes as low as 1500 with a normal differential count is a frequent finding of cardinal importance, but a normal leukocyte count does not exclude the diagnosis. A total eosinophil count not infrequently reveals an eosinopenia. *In active cases this may be sufficiently low to be an important diagnostic aid.* Moderate thrombopenia and hypochromic anemia often co-exist and represent a general depression of bone marrow function. However, a leukocytosis may occur in response to an intercurrent infection.

The L.E. cells, described by Hargraves *et al.*[11] in 1948 have provided a most useful diagnostic aid. These cells occur in rosettes as clumped polymorphs and macrophages which contain inclusion bodies. (Fig. 202.) This abnormal configuration is due to altered properties in the serum and

to occur in the viscera rather than in the skin and locomotor as systems in scleroderma.

Contrary evidence in favor of a primary disturbance in calcium metabolism is suggested by the production of sclerodermatoid lesions in young rats following parathyroid hormone administration.[43] Also this is suggested by the massive calcium deposits observed after prolonged, excessive doses



FIG. 208.—Hand of an adolescent white girl suffering from mild, generalized scleroderma of about five years duration. Note the circumscribed lesions of calcinosis in the skin of the second and fifth digits.



FIG. 209.—Sho[illegible]

can be reproduced by treating animal or normal human bone marrow with plasma from patients with disseminated lupus.

L.E. cells have been demonstrated in the peripheral blood or the bone *marrow in a high per cent of active cases. It* is necessary to count 500 to 1000 leukocytes before a negative report is acceptable. These cells are

other diseases.

Despite the above, it must be pointed out that when L E. cells are found in a typical case of rheumatoid arthritis, the question must be asked "does this prove that this case is really lupus?" Prolonged observation of the subsequent course of such cases will be necessary to answer this question In the meantime, an L. E. cell search should be made in every case of *"arthritis" in which systemic manifestations are prominent.*

The erythrocyte sedimentation rate is increased. The various serologic

of evoking specific antibodies in experimental animals. The abnormal globulins appear to be responsible for unusual transfusion reactions and for the development of agglutinins against the erythrocytes of all donors in certain phases of the disease, and are probably the cause of the spurious positive tests for lues as well as for the L.E effect. In the severe stages of

Azotemia occurs in terminal cases. The electrocardiogram may show low *voltage but is not characteristic. Blood cultures are sterile. Fluids from* the pleural, pericardial or peritoneal sacs are serofibrinous in character with a low leukocyte content. Seaman and Christerson[24] have recently detected L.E. cells in the pericardial and pleural fluid of cases of disseminated lupus erythematosus.

Differential Diagnosis.—The cardinal features are: a prolonged febrile illness with remissions, recurrent serous effusions, polyarthritis, photosensitivity, characteristic skin lesions and nephritis

Rheumatic fever, periarteritis nodosa, subacute bacterial endocarditis, rheumatoid arthritis and dermatomyositis are the main diseases likely to be confused.

From a practical point of view disseminated lupus should be considered as a possible diagnosis in any case of polyarthritis. The presence of leukopenia, hematuria and undue anxiety should heighten one's suspicion. Russell *et al*[25] report convulsions in 10 of 28 cases of disseminated lupus *They suggest that epilepsy occurring in a patient with arthritis* is presumptive evidence for lupus. An increased local or systemic reaction following exposure of a small area (3 x 3 inches) of skin to ultra-violet light may be

of vitamin D. These are roentgenographically indistinguishable from those of circumscribed calcinosis, but may disappear when vitamin D is withdrawn whereas those due to calcinosis are regrettably permanent.

Also, it seems rather unlikely that the large globular accretions of calcium, as seen in figure 210, represent merely the calcification of some abnormal matrix, since we have no evidence for the existence of such a matrix prior to occurrence of the calcification. Another explanation for these calcium deposits is a primary calcium-phosphorus retention, or failure of excretion, resulting in the continued deposition of calcium on an



Fig 210

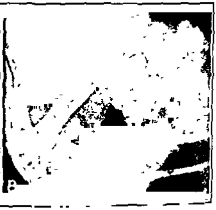


Fig 211

large joints

... kept for nineteen



... [illegible] of thorax of a guinea-pig kept on a diet deficient in greens

bone marrow is strong presumptive evidence for disseminated lupus erythematosus

Treatment.—There is no specific cure Extraction of infected teeth and tonsils, vaccine therapy, fever therapy, and heliotherapy are interdicted since they may cause an explosive flare-up with fatal results Bed rest, good nursing care, nutritious diet with supplementary vitamins, blood transfusions (but watch for reactions), avoidance of sunlight, and of all trauma are the basic features of management. Penicillin, streptomycin, iodine, liver extract, and testosterone have all been recommended but their value has not been established. Sulfonamides are not curative and may be

(PABA-Sodium) for long periods of time [31]*

Quinine was used long ago in the treatment of lupus More recently primaquin and chloroquin have been suggested in the rheumatic state, but have been ineffective in our hands. The most recent antimalarial to be advocated for lupus is atabrine in doses of 100 mg twice daily It is too early to make a statement as to the value of atabrine in this disease

Successful treatment must await a fuller understanding of the collagen diseases in general The fact that spontaneous remission occurs in about 25 per cent of cases gives reasonable hope that a method for producing artificial and perhaps permanent remissions will be discovered

About 150 cases of disseminated lupus erythematosus treated with cortisone and ACTH have been reported. There is general agreement that, as in all the collagen diseases, the active manifestations of the disease are strikingly suppressed, so that, from the viewpoint of the patient and his relatives, the improvement is dramatic There is also general agreement that these hormones neither affect the cause of, nor cure the disease, but may prolong the life of the patient. This is, as yet, unproved The suppressive effect of these agents conceivably could hold the disease in check until a spontaneous remission takes place ACTH, cortisone, and hydrocortisone are the only agents which have accomplished an effect of this magnitude and clinically they seem to be equally effective They are definitely indicated in the acute and sub-acute phases of the disease

Under adequate dosage, the fever, rash, arthritis, pleuritis, pericarditis, retinitis, Raynaud's phenomenon, mental disturbances as well as the abnormal electroencephalographic findings and acquired hemolytic anemia are all reversed Less constantly the anemia, abnormal serum proteins,

intramuscular injections or 200 to 300 mg of cortisone by mouth or paren-

* We have heard of several instances of hepatic necrosis incident to the use of this drug. Death may also occur from irreversible hypoglycemia (Ed)

originally microscopic focus It has been suggested that renal failure is the cause of calcium and phosphorus retention and it is true that renal lesions commonly occur in scleroderma However, frank renal failure is not a feature of the disease, and if the kidneys are a factor, their function must be selectively damaged. At any rate, there is still a place for the possibility of a disturbed calcium metabolism being of primary rather than secondary

A/G ratio[49] and a questionably increased serum calcium The histologic features of scleroderma were not present. This suggests, but does not prove, that a dietary deficiency may be one factor in the etiology of calcinosis and, by inference, scleroderma. Sclerodermatoid skin lesions have been described in severe malnutrition[23,32] Bernstein and Goldberger[7] report a complete reversal of scleroderma in a patient with presumed (but unproved) hypoparathyroidism following administration of dihydrotachysterol Their description of the appearance of the skin in this case seems

the affected tissues. The relation of the disturbed calcium metabolism to the disease is not understood, but opinion (unproved) to date favors the hypothesis that it is secondary to the fundamental process rather than a

l the[63]

the body there is induration of the affected tissues In the skin the earliest change is marked by non-pitting edema, subsequently by induration and finally by atrophy. All three stages may be present at the same time

The disease most commonly begins in the active decades of life but is not uncommon in children and has been reported in infancy and even in the new born[87] Occasionally, it coexists with rheumatoid arthritis, and sclerodermatoid swellings of the fingers are not uncommon in psoriatic arthritis (Figs 206 and 207)

In the "Shoulder-Hand Syndrome"[51] *which complicates 10 to 20 per cent* of cases of coronary occlusion, there may be painful swelling and stiffness of the hand with pigmented, waxy skin, Dupuytren's contractures on the

The lesions of scleroderma may undergo spontaneous arrest at any time although, naturally, those which have progressed to atrophy and replacement of parenchymal structures with fibrous tissue are irreversible. The

terally. When a good suppressive effect was achieved, the dose was tapered and the patient was maintained on a dose just sufficient to prevent a return of symptoms. This was usually 20 to 50 mg. of corticotropin and 50 to 100 mg. of cortisone per day. In some cases it was possible to discontinue therapy during remissions. Such a program represents an acceptable approach to therapy and is of the type generally recommended But even with the conservative program in which there was rigid sodium restriction and potassium supplements, many untoward effects due to the treatment were observed Congestive heart failure occurred in 10 per cent, hypertension in 9 per cent, mental aberrations in 4 per cent. Convulsions are not infrequent and may be due either to the disease or to hormonal therapy. In this series one-third of the patients died.

Dubois *et al.*[6] recommend a much more vigorous dosage. They gave 40 to 60 mg. of ACTH per day by slow intravenous drip and even as much as 600 mg. I V. in one day. Cortisone was given by mouth in doses from 150 to 500 mg daily for several days and even as much as 2400 mg. per day for three days was used in 1 patient in crisis. Their aim was to produce a state of Cushing's Syndrome, as judged by the moon face, as quickly as possible They regard this as the "end point" in determining dosage After it has been attained, the high dosage level is maintained for 1 or more weeks. Subsequently the patient is maintained on doses of the usual magnitude. (See Chapter 17.)

It is too early to decide on a standard technique of dosage in this disease, but it can be safely said that ACTH and cortisone, wisely used, offer an outstanding advance in the therapy of disseminated lupus erythematosus

OTHER POSSIBLE COLLAGEN DISEASES

[illegible] certain clinical fea-

[illegible]

Rheumatica

Erythema Nodosum.—This syndrome is said to be associated with a variety of infections of which tuberculosis and rheumatic fever are most commonly mentioned. (See Chapter 32) However, there is no real proof that any such specific etiology exists in the majority of cases. Drugs such as iodides and sulfonamides have been implicated in some cases. The disease may occur in localized epidemics which lends strong support to an infectious etiology. It often occurs sporadically however, and is most common in the spring of the year and in young people.

Well developed cases are characterized by a sore throat, malaise, fever and a mild general arthritis which may persist for some weeks but which leaves no permanent joint damage. The typical eruption occurs early in the course of the disease and consists of subcutaneous nodules, usually on the legs below the knees, which appear in crops at intervals of a few days. The nodules are raised, tender and seldom more than 2 inches in diameter. Their color varies from rose-pink to plum-color and the overlying skin is shiny and tense and often gives the appearance of impending suppuration which, however, never, or almost never occurs. As the lesions subside there is ecchymotic discoloration due to the extravasation of erythrocytes. X-ray of the lungs may show enlargement of the mediastinal lymph nodes.

author observed 1 case throughout her second pregnancy. In the first pregnancy, the patient observed a *partial* remission, in the second pregnancy no objective *improvement beyond* the ordinary variations of the disease were noted. As the disease gradually progresses there is ...
weakness
tion—us
pulmona



Fig. 213 —Case of L F., white female, aged sixty-two who had suffered from scleroderma for thirty-five years Note the dilated esophagus and retention of barium mixture at the lower end, also the cloud-like calcium deposits of circumscribed calcinosis in the skin and at spinal articulations

Symptoms.—The onset is usually insidious but occasionally may be sudden with widespread involvement of the skin following an acute infection. There may be prodromal paresthesias, arthralgias, and clinically

deformities of the late stage of the disease As the disease progresses dyspnea due to pulmonary fibrosis and to myocardial, and, occasionally valvular, sclerosis is a common complaint. Late in the disease breathing may be further hampered by a tight, adherent skin over the thorax which

There is no recognized specific treatment for erythema nodosum. The disease is self-limiting and benign Salicylates may be used for palliation. In 1951, three single case reports[1,7,28] of the effect of cortisone on erythema nodosum are noted In each case there was suppression of the clinical manifestations of the disease This does not prove that cortisone is indicated as routine treatment

Purpura Rheumatica (Schonlein's Disease) —This syndrome is a manifestation of idiopathic or anaphylactoid purpura When the purpuric skin lesions are uncomplicated the term *purpura simplex* is used When bowel and kidney lesions occur the term *Henoch's Purpura* may be applied. When articular lesions occur the disease may be referred to as *Schonlein's Purpura* or *Purpura Rheumatica* The etiology is unsettled Infection may precede

blood vessels

Idiopathic purpura occurs most often in children and young adults and is generally characterized by recurrent episodes which last for days or

combinations of the above signs and symptoms The polyarthritis is transient and leaves no residual joint damage The intestinal symptoms consist of crampy abdominal pain and melena and the renal picture resembles that of acute nephritis. The prognosis is good

suppressed but relapses occurred after stopping treatment and the course of the disease was not fundamentally altered

BIBLIOGRAPHY

1 ALBRIGHT, R W, and KUFFEL, M J · California Med, 75, 368, 1951
2 BAGGENSTOSS, A H, SHICK, R M, and POLLEY, H F Am Jour Path, 27, 537, 1951
3 BARDEN, R. P, and COOPER, D A Radiology, 51, 44, 1948
4 CLUXTON, H E, JR., and KRAUSE, L A M Ann Int Med, 19, 843, 1943
4a CURTIS, A C, BLAYLOCK, H C, and HARRELL, E R JR Jour Am Med Assn, 150, 844, 1952
5
6
7
8
9
10
11 Proc Staff Meet, Mayo

limits normal respiratory motion The patient, even early in the course of the disease may also complain of dysphagia. At first he notes inability to swallow more than a few mouthfuls in rapid succession There may be epigastric and lower retrosternal distress immediately on eating due to

of the whole esophagus so that gravity is necessary to assist the passage of food into the stomach.

Physical Findings.—The skin of the head, neck and upper extremities is most commonly affected, with lesser emphasis on the torso and lower extremities. However many exceptions to this distribution occur Among these may be mentioned the localized patches known as morphea and rare instances of hemi-scleroderma in which the affected area seemed to follow the distribution of peripheral nerves[50] A form beginning in the fingers

(Figs. 209 and 213) so that we see no reason to consider it a separate disease.

Features of Scleroderma Which Aid in Distinguishing It from Other Members of the Collagen Group of Diseases

1. Prodromal Raynaud's phenomena in fingers is common.
2. Skin changes are outstanding, with edema, induration, marginal inflammation, loss of hair, pigmentary changes and later atrophy and fibrosis with adherence to underlying structures
3. Skin and muscle contractures causing joint deformity Arthritis, with loss of cartilage
4. Dysphagia (patient may have to sit up to swallow)
5. Dyspnoea—due to pulmonary fibrosis (Rarely associated with traction bronchiectasis)
6. Roentgenograms may show Co-existing calcinosis, concentric bone atrophy of digits, lesions of peridental membrane, pulmonary fibrosis, loss of normal peristaltic pattern in esophagus and G I tract
7. Calcium balance shows calcium retention even without demonstrable calcinosis
8. Sedimentation rate generally normal

The affected skin is at first thickened (Fig 203) and appears as a non-pitting area of edema This phase is sometimes absent. It is followed by induration so that the skin cannot be pinched or picked up Later, the skin and subcutaneous tissues are all firmly bound to the underlying structures and motion is limited by fibrosis (Fig 204) The affected areas become hard and inelastic with various combinations of increased or decreased pigmentation, loss of hair and sometimes erythema at the margins In many cases there is a diffuse yellowish brown pigmentation As the stage of atrophy is reached the skin and subcutaneous tissues are united in a thin parchment-like structure which is stretched over the bones and atrophic

12 KAPOSI, M. K.: Arch f Dermat. u Syph., 4, 36, 1872
13. KEEFER, C. S , and FELTY, A. R Bull. Johns Hopkins Hosp , 35, 294, 1924
14. KLEMPERER, P.· Ann Int Med , 28, 1, 1948.
15 KUSSMAUL, A , and MAIER, R.· Deutsch Arch f. klin Med , 1, 484, 1866
15a LANSBURY, J to be published
16. LEVINSON, J E., HORWITZ, M , KULKA, J. R , PAGE, L , and BAUER, W.: Ann. Rheum Dis , 10, 255, 1951.
17. LOVSHIN, LEONARD L Proc Staff Meet , Mayo Clinic, 24, 48, 1949
18. OSLER, W : Am Jour Med Sci , 110, 629, 1895
19 POLLEY, H F , CLAGETT, O T , McDONALD, J. R , and SCHMIDT, H W.: Presented at the Annual Meeting of the American Rheumatism Association, June 7, 1952
20 RICH, A. R Bull. Johns Hopkins Hosp , 71, 123, 1942.
21. ——— Ibid , 71, 375, 1942
22. ———· Ibid , 77, 43, 1945
23. RUSSELL, P W , HASERICK, J R , and ZUCKER, E M.: Arch Int. Med , 88, 78, 1951
24. SEAMAN, A J , and CHRISTERSON, J. W.: Jour. Am. Med. Assn , 149, 145, 1952
25. [illegible]
26 [illegible]
27. [illegible]
28 [illegible]
29 [illegible]
30. YARDUMIAN, K., and KLEINERMAN, J.· Arch Int. Med , 83, 1, 1949.
31 ZARAFONETIS, C J. D , CURTIS, A. C., and GREKIN, R. H.: Univ. Hosp Bull (University of Michigan), 13, 122, 1947.

muscles. At this stage the face is small and expressionless with pinched nose, thin, gaping mouth, and possibly ectropion which together give a mummified appearance.

Similar changes occur in the extremities which give rise to various types of contractures and limitation of joint motion. (Fig. 204.) These need seldom be confused with arthritis since the characteristic fusiform enlarge-

membrane consisting of depressed white areas have already been mentioned.[4] We have observed corneal vascularization in 1 case

Esophagoscopic examination[26] may show thickened walls with smooth, white exudate in the lower one-third which on peeling leaves a granulating base.

the hands may show quite early a concentric diminution in the volume of the phalangeal bones. This may be extreme and lead to absorption, sometimes without pain. (Fig. 205.) Calcifications are frequently found if looked for, and vary from minute spicules in or under the skin to huge globular *collections situated deep in the region of the large joints.* (Fig 210) Dif-

This group of calcifica-
cinosis.
es reveals striking
own pathologic and

changes may be demonstrated in the small bowel and colon [6,36,41] Diffuse pulmonary fibrosis (Fig. 214), most marked in the lower portions of the lungs is not an uncommon finding. Dental x-rays have been reported[45] showing widening of the periodontal membrane in an even manner around the tooth root which is sufficiently characteristic to confirm an otherwise

calcium, phosphorus and phosphatase, basal metabolism, and ordinarily available routine blood chemistry determinations are normal. The positive calcium balance has already been mentioned. Creatinuria,[11] *diminished urinary 17 ketosteroids,* [27] and minor deviations in the lymphocyte count reported but are not of
od.
ma is not fusiform as in
due to stiffness rather
id the pulmonary and
alimentary lesions of scleroderma clearly distinguish it from rheumatoid arthritis. The thickened integument seen in the fingers of psoriatic

CHAPTER 37

THE SCLERODERMA GROUP OF COLLAGEN DISEASES

(Scleroderma, Sclerodactylia, Dermatomyositis and Calcinosis)

By John Lansbury, M D

Scleroderma and allied states are commonly mistaken for arthritis

system.



Fig 203 —Hands of a middle-aged white woman showing the edematous and indurative stage of scleroderma without atrophy The disease had been present for many years and the main complaint was severe dyspnea due to far-advanced pulmonary fibrosis of sclerodermatous etiology.

We are concerned here ...

... lesions of the shoulder-hand syndrome and of psoriatic arthritis The reader is referred to standard dermatology texts for a description of the patchy sclerodermatous lesions known as morphea, card and white-spot disease and scleroderma en coup de sabre.

acrosclerosis or the subsequent he former The id syndrome are ytren's contracma, but the skin accompanied by anes-*oma en cuirasse* may the breast in females ly be distinguished by biopsy.



FIG 214 —Case of scleroderma of many years' duration showing far advanced pulmonary fibrosis, most marked at the bases, and circumscribed calcinosis around the humeral heads and in the skin of the thorax.

Dermatomyositis has many features in common with scleroderma but differs mainly in the severity of the muscular weakness and atrophy. **Scleredema** generally follows an acute infection and is not to be confused with scleroderma In scleredema the skin is the site of a non-pitting edema due to thickening of the collagen bundles The skin is not bound down and there are no vascular changes, atrophy or calcification The disease is self limited, involutes in three to twelve months and seldom involves the digits Hematrophy of the subcutaneous tissues has often been described as scleroderma, but the skin is not bound down and, therefore, it is probably not true scleroderma

SCLERODERMA

Morbid
the colla
tion witl
fat is re
tissues are fragmented, disordered or absent and the arterioles are oblit

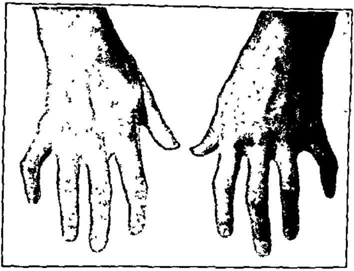


Fig 204 —Hands of an adolescent white girl who had had rather mild, diffuse scleroderma since the age of nine. Note the flexion deformities at the proximal interphalange joints and the characteristic, small, chronic ulcerations on the knuckles some of whic contained calcium granules The palmar surfaces of the hands show several areas circumscribed calcinosis (Fig 208)

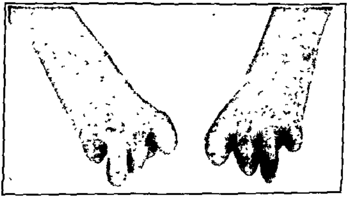


Fig 205 —Hands of a white female, aged sixty-two, showing extreme degree of

and cough which were due to the visceral lesions of scleroderma in the lungs and esophagus as seen in figures 213 and 214

Treatment.—The large number of unrelated agents recommended in the treatment of scleroderma is proof of their ineffectiveness. There is, at present, no cure for scleroderma and, unfortunately, no reliable palliative treatment.

eff

sur

v vascular. Thus, sympathectomy while vasodilation in the head and extremities, obviously cannot overcome the lesions of the heart, lungs and alimentary canal and, therefore, should be abandoned, except possibly in rare early cases where Raynaud's phenomenon demands relief. Our experience with general vasodilators has not been encouraging. Local vasodilation by mecholyl iontophoresis has been persistently advocated,[16] but has not come into general use.

Zarafonetis[56] has observed about 40 cases of scleroderma under long range treatment with potassium para-amino benzoate. In cases of moderate severity he noted a consistent softening of the skin, and believes that PABA therapy is superior to cortisone or ACTH for this disease. The improvement takes place slowly. Administration of 10 to 12 Gm. of the drug in divided doses each day is continued indefinitely. Patients must be warned not to refrain from eating while on the drug, since irreversible hypoglycemia occasionally occurs on fasting. Naturally, the fibrous contractures from the disease cannot respond to any medication. The aim of treatment must be to arrest the edematous and inflammatory phase of the disease

Of therapies aimed at altering the basic but unknown cause of the disease, the vitamins and hormones have followed much the same paths as they have in the treatment of rheumatoid arthritis Although one may occasionally find evidences for deficiency of vitamins of the B group and of vitamin C, these are incidental and treatment by them does not alter the course of the disease. Claims of benefit with large doses of vitamin D have not been confirmed and theoretically seem unsound since hypervitaminosis D causes calcific deposits indistinguishable from those of the calcinosis which not uncommonly accompanies scleroderma. Local applications of cod liver oil to the skin are still used by dermatologists but have not benefited those cases which have come under our observation Treatment with the "anti-stiffness factor"* which is presumably a vitamin, has given, in our experience, inconsistent results.[59] We have observed some improvement in 2 cases following a diet low in calcium and phosphorus

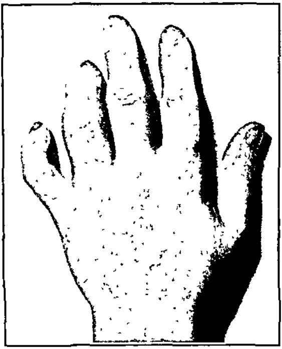
Most of the commercially available hormones have been advocated at some time or another Thyroid substance while capable of reversing the

common feature of scleroderma. On account of the disturbed calcium metabolism in scleroderma, parathyroidectomy has been undertaken although these glands have generally been found histologically normal in the few cases where they have been studied. Scleroderma has been reported

*Kindly supplied by Eli Lilly Co.

ated by connective tissue proliferation in the muscularis The epidermis is atrophic. The capillary pattern observed in life at the nail beds differs from that of Raynaud's Disease.[10] In the fingers the fibrosis impairs blood supply, and, by continuous pressure causes concentric bone atrophy resulting in various degrees of absorption of the digits (Fig. 205)

The visceral lesions of scleroderma may be summarized as follows:—the alimentary canal from lips to anus may be involved. Depressed areas of whitened mucous membrane have been described on the lips[4] and widening



[illegible]

digits

of the periodontal membrane[45] The muscular layer of the esophagus, stomach, small bowel and colon is replaced by fibrous tissue with resulting impairment in motor function, but the nervous plexuses of Auerbach remain intact Lower esophageal obstruction is due to spasm and not to stenosis[6] The myocardium is infiltrated with focal overgrowths of vascularized connective tissue which replace muscle fibers and which may be extensive enough to cause myocardial failure.[27 31] A contributing factor to this is pulmonary fibrosis in which alveoli are replaced by vascularized connective tissue Atrophy of the uterus and prostate have been noted, also fibrosis and atrophy of the thyroid gland. The pituitary, parathyroids,

in the presence of hypoparathyroidism [7] The claims of benefit from parathyroidectomy have not been confirmed, and, like sympathectomy, this procedure has been given up.

Posterior pituitary extract, parathyroid extract, estrogens and testosterone have all been advocated with claims of improvement in the skin, ... the disease. ... een tried and found ... is have seemed to produce benefits in sporadic cases. An interesting report by O'Leary[33] claims recession of edematous swelling in 7 of 9 patients following benadryl ...

dilation may be tried

As would be expected on theoretical grounds, ACTH and cortisone administration suppresses the active inflammatory and edematous phases of scleroderma. They do not, however, alter the fundamental course of the disease, nor rectify the damage caused by fibrosis, scarring and contra...

not too encouraging.

DERMATOMYOSITIS

Clinical Features —Dermatomyositis is a rare disease In its later stages it is not uncommonly mistaken for rheumatoid arthritis on account of the joint deformities and fixations which, however, are due to muscular contractions and fibrosis of skin and fascia, and not to disease of the joints themselves The disease has many features in common with scleroderma and histopathologically belongs to the collagen necrosis group of diseases We shall present below a brief outline of its clinical and pathological features

Etiology and Pathology.—The etiology is unknown, but the general clinical manifestations of the disease suggest an abnormal reaction to inf...

have been advanced as explanations for the etiology of dermatomyositis and with equally negative results

Histologic study of the muscles shows degeneration of the parenchymal elements with some attempt at repair as judged by proliferation of muscle nuclei There is an invasion by lymphocytes, histiocytes, plasma cells and fibroblasts. ...
no sclerosis of
damage, are
dermatomyos
point,[8] and certain observers[14] claim that the histopathology is essentially the same in the two diseases. From a clinical viewpoint, the involvement of the skin, muscles and heart, and the frequent coexistence of calcinosis

adrenals, ovaries and testes are not significantly affected. The kidneys show *fibrinoid degeneration in the vascular walls and glomerular lesions* similar to those of disseminated lupus.

Deposits of calcium salts (phosphate and carbonate) in the skin, subcutaneous tissues, muscles and fascial planes and at the articulations are common and are frequently overlooked unless a roentgen survey is made especially for this purpose.

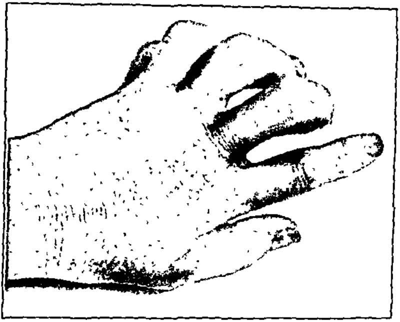


Fig. 207.—For comparison with figure 206 showing rapid advance of sclerodermatoid lesions of fingers in psoriatic arthritis.

Etiology.—*The etiology of* scleroderma is unknown. Females are more commonly affected than males. The onset may be at any age. There is no proof of endocrine causation. Reduction in the swelling following antihistaminics[33] suggests an allergic factor. Jadassohn showed by histologic studies that calcium is deposited diffusely between the collagen fibers of the corium[29] and *Leriche and Jung demonstrated by chemical methods an*

series of calcium balances using dummy meals to control the calcium intake over adequate periods of time, found calcium and phosphorus retention in cases of scleroderma. They concluded that the damaged collagen had an affinity for calcium and that a primary disturbance in calcium metabolism had not been proved. Their hypothesis is further supported by the normal serum calcium and phosphorus values in cases of scleroderma and calcinosis and by the known affinity of gels for calcium as shown by Amberg and Lansbury.[1] There is no evidence for hyperparathyroidism in scleroderma or calcinosis. The metastatic calcifications due to hyperparathyroidism tend

are features common to both the diseases, whereas the pulmonary and alimentary tract lesions, and the principal involvement of the skin rather than the muscles, which characterize scleroderma, are sufficient to differentiate the two diseases.

The heart in dermatomyositis may show lesions in the myocardium

The onset may be at any age but occurrence in the middle decades is more

In many cases an acute infection such as a sore throat antedates the onset. The disease may evolve rapidly and run a fulminating course, but, more commonly, the onset is gradual with vague prodromal symptoms such as malaise, weakness and muscle aches In the active phases the

Edema is the most chara
tion is variable and unrelated to the
extremities are commonly affected and, on account of orbital involvement, the appearance of the patient may resemble that of trichinosis. Of interest
changes in color and texture which may
atrophicans vasculare,
se may be superimposed
e disease progresses, the
in contrast

scleroderma and serves to differentiate the two diseases

The *musc*
and pain on
account for
The affected muscles may be *tender* on pressure, and, as the
gresses they may feel doughy, and eventually firm and even board-like. In this latter stage there are frequently contractures, generally symmetrical, which cause fixation of joints and render the patient helpless. There is an early predilection for the muscles of the shoulder and pelvic girdles, and involvement of the neck and shoulders may give rise to a characteristic posture with shoulders falling forward and head drooping Involvement of

the musculature of the orbit may cause diplopia, of the larynx may cause hoarseness, of the face may cause weakness resembling that of myasthenia

[illegible]

diffusely throughout the musculature (Fig 215.)



[illegible]

calcinosis

General features include stomatitis, fever, splenomegaly, lymphadenopathy, hepatomegaly, weight loss, Raynaud's phenomenon, paresthesias, sweating, and cardiac enlargement These features are by no means constant and occurred sporadically in a group of 40 cases analyzed by O'Leary and Waisman [44] Rheumatoid arthritis coexisted in 2 of the 40 cases but

PART VI

DEGENERATIVE JOINT DISEASE (OSTEOARTHRITIS)

CHAPTER 38

ETIOLOGY AND PATHOGENESIS OF DEGENERATIVE JOINT DISEASE

By NATHAN R ABRAMS, M D.

Definition — *Degenerative joint disease* (syn.. osteo-arthritis, hypertrophic

in joint diseases made, until the nineteenth century when observations on postmortem material and on living patients were reported.

In 1824, Bell[11] described eburnation of the femoral head Soon afterward, (1835), Robert Smith[50] proposed the term *"morbus coxae senilis"* for

information when he described the gross and microscopic changes of the disease It was his belief that this condition was related to the physiologic process of aging Many other important contributions followed, [10 12,42 45 52 55] but it is the consensus among present day students of this disease that Weichselbaum's premise probably is correct in most cases

arthralgia was not uncommon. Calcinosis was observed in 5 *cases but not* all were subject to *x*-ray examination. Gangrene of the finger tips and exudates in the ocular fundi which resemble those seen in disseminated lupus, but [illegible]

Laborato [illegible]

O'Leary's s [illegible]

The white blood count is not characteristically altered, but a moderate increase in monocytes and eosinophiles is a common, but not a constant, finding. The urine shows a moderate amount of albumin and a few casts, indicating renal damage *Creatinuria,* which may vary greatly from day to *day, is present in excess.* Excretion of porphyrins was found normal The total serum *proteins were low but the albumin-globulin ratio* was unaltered Griffith[19] found the muscle utilization of glucose to be impaired and attributes this to failure to form glycogen.

It will be seen that none of these laboratory findings is diagnostic. The absence of leucopenia may be of assistance in excluding lupus erythematosis. The creatinuria is suggestive evidence in favor of dermatomyositis, but since it roughly parallels the severity of the disease, it is of least value when it is most needed.

Differential Diagnosis.—The well-established case presents no serious problem in diagnosis. In the earlier phases it may be difficult to distinguish from scleredema or scleroderma. The diffuse muscular pain and weakness is the main distinguishing point The facial swelling of the acute phase is differentiated from that of trichinosis by the epidemiologic background, gastrointestinal symptoms and the transient nature of the facial swelling in trichinosis

The clinical picture of early lupus erythematosis may be indistinguishable from mild cases of dermatomyositis. The absence of muscular lesions in lupus on biopsy may aid in the diagnosis.

Dermatomyositis

Features Which Aid in Distinguishing Dermatomyositis from Other Members of the Collagen Group of Diseases

1. Muscle weakness and atrophy are the outstanding features
2. *Creatinuria may occur with rapid muscular wasting*
3. [illegible] in about 15% of cases
4. [illegible]
5. [illegible] " of the collagen diseases *and presents many features resembling scleroderma* and disseminated lupus erythematosus

Also to be considered in the differential diagnosis are the *following:*—pellagra, myasthemia gravis, acute rheumatic fever, periarteritis nodosa and the myopathies

Treatment.—Zarafonetis[56], in a personal communication, recommends the administration of PABA (potassium para-amino benzoate) for dermatomyositis. He observed results in twelve cases which have seemed to parallel those reported for scleroderma.

11. Craig, J, and Lyall, A.: Brit Jour Child. Dis, 28, 29, 1931
12. Cornbleet and Struck. Arch. Derm and Syph, 35, 188, 1937.
13. Curtis, L. E, and Feller, A E · Ann. Int. Med, 17, 1005, 1942
14 Dowling, G B, and Griffiths, W. J · *Lancet*, 1, *1424*, *1939*.
15. Durham, R H.· Arch Int Med, 42, 467, 1928.
16. Duryee, A W, and Wright, I S. Am. Heart Jour, 14, 603, 1937.
17 Epstein, E, and Ayres, S, Jr: Jour Lab and Clin. Med, 22, 697, 1937
18 Friedlander, J: Deut Arch f kiln Med, 166, 107, 1930
19. Griffith, W J Brit Jour Dermat. and Syph, 52, 295, 1940
20 Jadossohn, J.· Arch f Dermat u. Syph, 126, 172, 1919.
21. Keil, H. Ann. Int Med., 16, 828, 1942
22 Kennedy, R. L J. Proc. Staff Meet Mayo Clin, 7, 329, 1932.
23. Langmead, F. S Arch. Pediat, 40, 112, 1923.
24 Leriche, R and Jung, A Bull. Soc. Franz de Dermat d Syph, 42, 885, 1935
25. Ligman, J V Arch of Ophth, 37, 155, 1947.
26 Lindsay, J. R, Templeton, F. E, and Rothman, S, Jour Am Med. Assn, 123, 745, 1943
27. Mathisen, A K, Palmer, J. D: Am Heart Jour., 33, 366, 1947.
28. McMillan, R B Brit. Med Jour, 2, 229, 1943.
29. Medvei, V C: Lancet, 2, 708, 1945
30. Millharat, A T, Toscani, V, and Bartels, W. E: *Proc Soc. Exper Biol and* Med., 58, 40, 1945
31 Murphy, J B, Krainin, R., and Gerson, M J.: Jour. Am Med. Assn, 116, 499, 1941
32. Naville, M [illegible]
33 O'Leary, P [illegible]
34. O'Leary, P [illegible] 1940
35 Pedersen, J [illegible]
36 Rake, Geoff [illegible]
37. Rodrigriez [illegible], 3074, 1945
[illegible] Acta Med Scand, 100, 57, 1939
[illegible]
30, 25, 1944
46. Steinbrocker, O, Spitzer, N, and Friedman, H. H. Ann Int Med, 29, 22, 1948
47. [illegible] Proc Soc Exp Biol and Med, 30, 920, 1933
48 [illegible] of Pediatr, 53, 550, 1936
49. [illegible] Jour. Biol. Chem, 159, 247, 1945
50 [illegible]1, 1945.
51. [illegible], and Bailey, O. T: Arch Int Med, [illegible]
[illegible] Arch Int Med, 42, 483, 1928
[illegible]936
[illegible] Int. Med, 85, 27, 1950.
57. Andrews, G C Arch. *Derm* and *Syph*, *64*, *218*, *1951*.
58 Hines, E. A, Wakin, K. G., Roth, G M, and Kierland, R R: Jour. Lab and Clin Med, 96, 834, 1950
59 Lansbury, J., Smith, L W., Wulzen, R, and VanWagtendonk, W. J.· Ann Rheum Dis, 9, 97, 1950.
60. Oppel, T W, Coper, C., and Michorat, A. T.: Ann. Int. Med, 32, 318, 1950
61. Taubenhaus, M, and Lev, M.: Arch Int. Med, 87, 583, 1951.
62 Thorn, G W., *et al* · New England Jour Med, 242, 824, 1950.
63. Wheeler, C. E., Curtis, A. C., Cawley, E. P., Grekin, R. H, and Zheutlin, B · Ann. Int Med, 36, 1050, 1952.

ACTH and cortisone therapy has so far been reported in too few cases for final appraisal, but at present seems to offer the most potent and worthwhile form of treatment Thorn *et al.*,[670] Oppel,[60] and Andrews[57] all observed improvement in dermatomyositis with ACTH administration Oppel recorded a two-month remission following a course of fifty-three days of treatment with ACTH.

General hygienic measures and the use of a respirator and tube feeding in the terminal phases are obviously indicated though ultimately ineffective O'Leary[34] recommends removal of gross foci of infection, fever therapy and oxygen inhalations if the patient is not mortally ill A variety of other agents, following the general pattern of those tried in scleroderma have been advocated with no proof of real value Wheat germ oil and soy bean lecithin may cause a reduction in the creatinine excretion in dermatomyositis and progressive muscular dystrophy,[30] but do not improve the clinical symptoms

CALCINOSIS

Calcinosis (sometimes called calcium gout or chalk gout) is a rare disease which occurs in 2 forms known respectively as *circumscribed* and *interstitial calcinosis* Few observers have had the opportunity to study more than 1 case—a fact which is proved by the continuing half-dozen or so single case reports from various parts of the world which still appear annually, although the disease was described about seventy years ago Several good

Etiology.—As noted in the previous chapters, calcium retention and deposition in the connective tissues, but not in the parenchyma, is a feature

routine roentgen surveys were made on all cases of scleroderma and dermatomyositis, the deep calcinotic lesions would be more frequently reported

The significance of this inter-relationship is not understood Langmead,[23] on clinical grounds, suggested that all these syndromes were manifestations of a single disease Some have questioned the independent existence of calcinosis and have regarded it as merely a complication of scleroderma and dermatomyositis. The prevailing viewpoint accepts the syndromes as individual diseases, and recognizes but does not explain the several features which are common to them

Pathology.—The histologic studies of calcinosis are not sufficiently numerous to permit a final conclusion as to whether it is a separate disease or a complication of scleroderma and dermatomyositis Observers have reported degeneration of fatty and connective tissue, chronic inflammation of connective tissues, skin atrophy, interstitial myositis, degeneration of muscle fibres and perivascular infiltration But, considered in toto, these

PART VI

DEGENERATIVE JOINT DISEASE (OSTEOARTHRITIS)

CHAPTER 38

ETIOLOGY AND PATHOGENESIS OF DEGENERATIVE JOINT DISEASE

By Nathan R Abrams, M D.

Definition.— *Degenerative joint disease* (syn.· osteo-arthritis, hypertrophic arthritis, degenerative arthritis, arthritis deformans, senescent arthritis)

(500,000 years ago). Probably the oldest known human case is that of the Neanderthal man (± 40,000 years B C), who had characteristic changes in

in joint diseases made, until the nineteenth century when observations on postmortem material and on living patients were reported.

In 1824, Bell[11] described eburnation of the femoral head Soon afterward, (1835), Robert Smith[59] proposed the term *"morbus coxae senilis"* for

information when he described the gross and microscopic changes of the disease. It was his belief that this condition was related to the physiologic process of aging. Many other important contributions followed,[10,12 42 45 52,55] but it is the consensus among present day students of this disease that Weichselbaum's premise probably is correct in most cases

changes can hardly be regarded as the specific manifestations of calcinosis since they are similar to, if not identical with, the histologic changes seen in



FIG. 216.—Roentgenogram showing hypodermoliths in the region of the buttock. The etiology of hypodermoliths is unknown but is thought in many cases to be due to intramuscular injections of radio-opaque material. They are *unrelated* to *calcinosis*. Note the phlebolith just above the ramus of the pubis.

Disturbed Physiology of Calcinosis.—Friedlander[18] reported in 1930 normal balances between intake and output of nitrogen, sodium, potassium, magnesium, calcium and phosphorus in calcinosis. Pedersen[35] noted the coexistence of true gout and a *diminished* output of *urinary androgens* in

there was an elevation of blood uric acid. These, we note, were in some cases minimal and, on the whole, might be explained on the basis of impaired uric acid excretion. There is no evidence that true gout is in any way related to calcinosis.

The calcium deposits on analysis[52,53] consist mainly of calcium phosphate, with small amounts of carbonate, and traces of magnesium. The serum

More recent work include
servations of various joints
of fifteen and ninety-five
were found in the knee joint
and microscopic appearances of 100 knee joints from 77 consecutive necropsies.[47] They found that 66 per cent of the joints in individuals between thirty and thirty-nine, and over 90 per cent of all knee joints in patients over forty showed degenerative changes They concluded that *degenerative arthritis is a process associated with the aging of the tissues of joints, aggravated by strain, hemorrhage, trauma, and static deformities, the end results depending on a summation of these factors.*

Bennett, Waine, and Bauer studied the knee joints of 63 individuals varying in age from one month to ninety years and having no history of joint pain,[11] these joints were obtained either postmortem or after amputation.

the second decade of life and increased in frequency and severity with advancing age.

In a study of the reparative ability of cartilage, Bauer and Bennett produced changes characteristic of human degenerative arthritis by displacing the patella in rabbits without entering or injuring the knee joints,[12] and in dogs by creating defects surgically in the articular cartilage after patellar displacement [8] Changes were observed as early as four weeks after beginning the experiment Thus, they concluded that *degenerative arthritis is the result of the wear and tear of increasing age and repeated trauma which might be caused in a variety of ways. Because the changes were degenerative in nature, they challenged the use of the word "arthritis" proposing the term "degenerative joint disease" in its place* This term has been accepted by the American Rheumatism Association and will be used throughout this chapter

Unfortunately not all cases can be explained on this basis, for many individuals with marked changes in their joints have no symptoms while others with minimal pathological alterations are afflicted with symptoms of the disease [7] It has been stated that degenerative joint disease is present universally after the age of forty, but only about 5 per cent of the people are clinical victims of the disease.[31] The theory does not explain why certain joints such as the terminal interphalangeal joints of the fingers commonly are affected while many other joints rarely are involved.

For these reasons, numerous other theories have been advanced to explain the etiology of this disease. These have included endocrine dysfunction, chronic vascular disease with resultant local anemia; focal infection; metabolic disorders; aseptic necrosis of bone and/or cartilage, physiologic stress and strain, heredity, aging and trauma Most of these theories have been discarded but some undoubtedly are partial factors in the explanation of certain individual cases

pational trauma was the most important single causative factor in the etiology of the disease. This idea was refuted, however, by the work of

calcium and phosphorus are generally normal, although in some cases the calcium is elevated. A calcium balance study by Bauer[5] showed retention of calcium with a lesser retention of phosphorus Since the main feature of calcinosis is retention and deposition of calcium phosphates there can be no

would reveal minor deviations from normal renal function have been undertaken. Studies of adrenal cortical function should be done

Pathogenesis.—The cause of calcinosis is unknown Its close association with Raynaud's phenomena, dermatomyositis and scleroderma has already been noted but sheds no light on the nature or cause of the disease. An important controversy exists as to whether calcinosis is due to a primary

lesion consists of damage to these tissues which confers upon them an avidity for calcium salts

In favor of the disturbance in calcium metabolism being primary are the very similar calcifications produced by excessive doses of vitamin D, (which

connective tissues without preceding necrosis, and, finally, the restoration to health with reported reversal of the skin and muscle lesions which follows

calcinosis, and that the skin and muscle lesions of scleroderma and dermatomyositis are not regularly reproduced in animals, although skin and muscle[55] lesions do occur. Also hyaline degeneration and fat necrosis have been reported to precede calcification by some observers [43]

The above remarks apply mainly to circumscribed calcinosis although some of the Wulzen animals (Fig 212) also showed the interstitial type of

The Clinical Picture.—*Circumscribed calcinosis* occurs about 6 times as frequently in females as in males and, in about a third of instances, is associated with Raynaud's phenomenon, sclerodactylia, or diffuse scleroderma. Adults are commonly affected and the disease has an insidious onset and mild course not incompatible with reasonably good health and long life. It may be detected by finding calcium deposits in the skin (Fig 208) which may or may not erupt, or, by careful palpation of the deep, rounded, nontender masses near the large joints. In some cases there are numerous nodules in the skin ranging in size from that of a pea to that of a small nut.

Heine[30] who, by autopsy material, compared the incidence of pathological joint changes in 100 individuals who had done hard labor for many years with an equal number of people of comparable age who had done little or no physical work. His results showed that there was no difference in the

abnormal mechanics of a joint resulting from chronic strain due to obesity and poor posture, contusions, dislocations or fractures near or into joints, malformations and malalignments of congenital, traumatic or neoplastic origin; and to damage resulting from infections, inflammatory or metabolic forms of joint disease.

While there is little doubt that physiologic stresses and strains cause

sites where functional stress is not present

(b) Aseptic Necrosis.—This theory was advanced when degenerative joint disease was observed after aseptic necrosis had developed in caisson workers[35] and in workers using pneumatic tools.[23, 41] Massive infarction of bone may result from caisson disease. If this necrotic bone borders on

"traumatic arthritis") due to such tools have appeared in the medical literature.

it is probable that physiological stress due to everyday use of a joint damaged by these diseases accounts for the appearance of such changes.

(d) Focal and Systemic Infection.—This theory has all but been discarded. No evidence of infection, either clinically or pathologically, has been demonstrated.

(e) Vascular Changes.—Because degenerative joint changes are present universally in the aged, this theory has appealed to many workers. However, most investigators who have studied the histologic appearance

The *overlying skin may become inflamed* especially over pressure points

may be seen deep in the wound. These usually heal slowly without much scarring. They may be evacuated *surgically*. *Their frequent occurrence* near joints has led to the term 'calcium gout' Calcium may also occur as 'sand' in the skin, or in plaques beneath the skin. The circumscribed *masses are commonly found* in the fingers and near the small joints of the hand.

These deposits cause little real disability except local pain and tenderness. They do not of themselves cause crippling, and may be overshadowed by the coexistent scleroderma.

The *serum calcium and phosphorus values are generally normal*, and

volume of a golf ball or a small orange. No deposits occur in the parenchymal organs. (Figs. 210, 213 and 214.)

Interstitial calcinosis, known also as universal or diffuse calcinosis, *presents quite a different picture. It commonly occurs in children or in* young adults, and has been reported at the age of two years An acute infection may precede it, and the first manifestation may be a scleredematous affection of the skin. This is accompanied by fever, malaise, and diffuse muscle pain and weakness—much as in the clinical picture of dermatomyositis. Later, there is weight loss, emaciation and atrophy of muscles and disappearance of subcutaneous fat. The skin is thickened, adherent to the underlying structures, and may contain numerous non-*tender, firm, discrete nodules* There is *great stiffness of the muscles* and ultimately limited motion of joints and deformity due to atrophy and fibrous contractures of the muscles. Splenomegaly has been reported

the disease.

X-rays show

es and fascial

of the interstitial connective tissue

The muscles are degenerated but

should be considered as primary

calcinosis, or as a form of dermatomyositis with intercurrent calcification of the diseased tissues is difficult to judge.

Diagnosis.—The diagnosis depends on recognition of cutaneous and subcutaneous calcium deposits, and is usually confirmed by *x*-ray. Metastatic calcifications due to chronic renal failure, vitamin *D intoxication,*

joint tissues are not inclined to consider arterial [illegible]
changes.[29,30,59] [illegible]
presence of ad [illegible]
Raynaud's Disease, particularly when these occur in young individuals Kling[33] found no relationship between vascular lesions in synovial vessels and the articular changes present, thus lending no support to the theory that degenerative joint disease results from degeneration secondary to arteriosclerosis of articular vessels.

(f) Endocrine Disturbances.—The relationship between endocrine disturbances and degenerative joint disease still is unsettled. In 1931, Erdheim[22] directed attention to the relationship of pituitary tumors and degenerative joint disease. Later, Waine, Bennett and Bauer[63] reported the pathological appearance of the joints in a case of advanced acromegaly. While the changes resembled those of degenerative joint disease in many ways certain differences were noted.

The occurrence of painful joints with the menopause has received attention by Cecil and Archer[17] and more recently by Hall[25] and Ishmael.[35] In most cases, patients in this category appeared to have arthralgia rather than true joint disease. However, it has been observed that degenerative joint disease frequently begins at this period

Some investigators have reported low basal metabolic rates in large percentages of patients with degenerative joint disease;[27,28] this has been refuted by others.[7,53,64]

(g) Heredity.—Stecher, in a study of 7000 individuals, reported a large hereditary factor in patients with Heberden nodes.[60] Many workers feel that the inheritance of poor grades of cartilage may be a factor in the premature appearance of degenerative joint disease.[8]

Summary of Etiologic Factors.—1. Practically all students of arthritis acknowledge the fact that *increased stresses and strains on joints (chronic irritation) comprise the most important causative factors in the production of degenerative joint disease.*

2. Not all cases can be attributed to this cause. Theories have been [illegible] focal [illegible]

appears to play a part. The rôle of endocrine disturbances has not been conclusively determined but probably is of minor importance.

NORMAL AGING OF JOINTS AND PATHOGENESIS OF DEGENERATIVE JOINT DISEASE

To understand degenerative joint disease, one must bear in mind the anatomical and histological make-up* of joints and in addition the *normal physiologic process of joint aging.* Most of our knowledge concerning the aging of joints has been gained from the studies of Rimann[55] (1906), Beitzke[10] (1912), Heine[30] (1926), Schmorl and Junghans[56] (1942), Parker, Keefer, Myers and Irwin[46,48] (1934), and Bennett, Waine and Bauer,[14] (1942).

A thorough review, study and discussion of degenerative joint disease and the process of joint aging was published recently by Bennett, Waine and

* Described in Chapters 3 and 4, pp. 42 and 48

nized hypervitaminosis D having been the cause of the calcification must be kept in mind since the lesions due to this may disappear spontaneously when the drug is withdrawn.

Treatment is aimed at removal of the abnormal calcium deposits. Attempts to mobilize calcium by use of parathyroid hormone, vitamin D or dihydrotachysterol are of little value because calcium is usually then mobilized from the skeleton rather than from the abnormal deposits, and, despite the increased calcium excretion that may be induced, further metastatic calcification may occur. However, acidification by ammonium chloride has been shown to increase calcium excretion and has resulted in some clinical improvement.[5] Kennedy[22] reported a case cured after three years treatment by a ketogenic diet used as an acidifying agent.

Another method of attack is reduction of calcium intake with the idea that the body will call on the abnormal calcium deposits to make up the deficiency. The large, well-vascularized skeleton competes for calcium with the relatively small foci of abnormal calcification. Craig and Lyall,[11] and others[8,48] report amelioration by this method with reduction in the amount of calcification as seen by x-ray as well as clinical improvement. Their treatment consists of a low calcium and phosphorus diet with 12 grams of alkaline sodium phosphate by mouth daily. The rationale of this treatment is the prevention of absorption of calcium from the intestinal tract. It is possible that the high fat content in the ketogenic diet may have a similar action by forming calcium soaps in the bowel.

A somewhat different approach was suggested by Snapper, and carried out by Roovers.[48] The case which he presents showed massive calcium deposits by x-ray and had multiple skin lesions and muscle contractures. An unusual feature was osteoporosis. No history of excess vitamin D ingestion was reported. The patient was given 4 gm daily of sodium citrate by mouth together with added calcium. Following this the serum calcium and phosphorus rose to above normal and the patient became seriously ill with a rectal temperature of 104°. After six months, the patient was completely well and roentgenograms showed disappearance of all calcification. A follow-up examination two years later found the patient perfectly well.

[illegible] this program

[illegible] violet irradiation or heliotherapy. Prolonged administration by the author of fat soluble concentrates from greens* which were capable of preventing the muscular lesions and calcific deposits in guinea pigs, on the deficient diet of Wulzen, failed to diminish the calcinotic lesions in humans.

BIBLIOGRAPHY

1 [illegible] 78, 47, 1928

[illegible] Dermat., 50, 267, 1938

[illegible] Jour. Med. Sci., 187, 237, 1931

7 Br[illegible], 130, 570, 1946
8 Br[illegible]
9 Br[illegible]
10 Br[illegible]

* Kindly supplied by Eli Lilly and Co.

Bauer.[11] These investigators reported the gross and microscopic appearances of knee joints obtained postmortem or following amputation in 63 individuals *None of their patients had complained of articular symptoms*



Fig. 217.—Normal right knee joint of child of 9 years. Cartilages and synovial [illegible]

and pale. At the points of attachment along the articular margins they merged into the hyaline cartilage. This tissue showed a gradual transition into denser, less vascular connective tissue, which in turn, as the midline area of the joint was reached, changed to fibrocartilage and finally to immature hyaline cartilage In older children, this layer became thicker and

* Figs 217-225 (Bennett, Waine, and Bauer, Courtesy of the Commonwealth Fund)

The overlying skin may become inflamed especially over pressure points such as knees and elbows, and the underlying calcific mass may suppurate The discharge may then contain calcium granules in gritty form, or a paste-like substance, and, occasionally solid white accumulations of calcium may be seen deep in the wound. These usually heal slowly without much scarring. They may be evacuated surgically. Their frequent occurrence near joints has led to the term 'calcium gout.' Calcium may also occur as 'sand' in the skin, or in plaques beneath the skin. The circumscribed masses are commonly found in the fingers and near the small joints of the hand

These deposits cause little real disability except local pain and tenderness They do not of themselves cause crippling, and may be overshadowed by the coexistent scleroderma.

The serum calcium and phosphorus values are generally normal, and [illegible]

enchymal organs. (Figs. 210, 213 and 214.)

Interstitial calcinosis, known also as universal or diffuse calcinosis, presents quite a different picture. It commonly occurs in children or in young adults, and has been reported at the age of two years An acute infection may precede it, and the first manifestation may be a scleredematous affection of the skin. This is accompanied by fever, malaise, and diffuse muscle pain and weakness—much as in the clinical picture of dermatomyositis. Later, there is weight loss, emaciation and atrophy of muscles and disappearance of subcutaneous fat. The skin is thickened, adherent to the underlying structures, and may contain numerous non-tender, firm, discrete nodules There is great stiffness of the muscles and ultimately limited motion of joints and deformity due to atrophy and fibrous contractures of the muscles Splenomegaly has been reported Recovery has been reported, but untreated cases may die of the disease

The serum calcium may be elevated, also the total protein. X-rays show widespread linear calcification of the subcutaneous tissues and fascial planes around and between the muscle fasciculi.

[illegible] of the interstitial connective tissue [illegible] The muscles are degenerated but [illegible] should be considered as primary calcinosis, or as a form of dermatomyositis with intercurrent calcification of the diseased tissues is difficult to judge.

Diagnosis.—The diagnosis depends on recognition of cutaneous and [illegible] Meta- [illegible] oxication. [illegible] the various [illegible] the fact [illegible] they are

more densely calcif . . .

lateral as well as longitudinal growth of the bony epiphysis occurred.

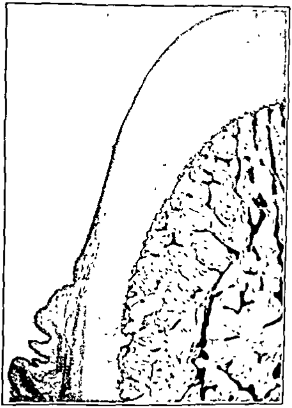


Fig 218.—Lateral margin of patellar surface of the femur × 6 Note that the synovial membrane merges gradually into the articular cartilage at the perichondrial margin Also note thickness of cartilage (same case as previous figure)



. . . × 23 Note the size . . . vial fibrous tissue is

nized hypervitaminosis D having been the cause of the calcification must be kept in mind since the lesions due to this may disappear spontaneously when the drug is withdrawn.

Treatment is aimed at removal of the abnormal calcium deposits Attempts to mobilize calcium by use of parathyroid hormone, vitamin D or dihydrotachysterol are of little value because calcium is usually then mobilized from the skeleton rather than from the abnormal deposits, and, despite the increased calcium excretion that may be induced, further metastatic calcification may occur. However, acidification by ammonium chloride has been shown to increase calcium excretion and has resulted in some clinical improvement.[5] Kennedy[22] reported a case cured after three years treatment by a ketogenic diet used as an acidifying agent

Another method of attack is reduction of calcium intake with the idea that the body will call on the abnormal calcium deposits to make up the deficiency. The large, well-vascularized skeleton competes for calcium with the relatively small foci of abnormal calcification Craig and Lyall,[11] and others[9,45] report amelioration by this method with reduction in the amount of calcification as seen by x-ray as well as clinical improvement Their treatment consists of a low calcium and phosphorus diet with 12 grams of alkaline sodium phosphate by mouth daily The rationale of this treatment is the prevention of absorption of calcium from the intestinal tract. It is possible that the high fat content in the ketogenic diet may have a similar action by forming calcium soaps in the bowel

A somewhat different approach was suggested by Snapper, and carried out by Roovers.[38] The case which he presents showed massive calcium deposits by x-ray and had multiple skin lesions and muscle contractures. An unusual feature was osteoporosis No history of excess vitamin D ingestion was reported The patient was given 4 gm daily of sodium citrate by mouth together with added calcium Following this the serum calcium and phosphorus rose to above normal and the patient became seriously ill with a rectal temperature of 104° After six months, the patient was completely well and roentgenograms showed disappearance of all calcification A follow-up examination two years later found the patient perfectly well

It is difficult to interpret this report, but a further trial of this program would be interesting

Various other therapies have been advocated such as ultra-violet irradiation or heliotherapy. Prolonged administration by the author of fat [illegible]

BIBLIOGRAPHY

1 Amberg, S, and Lansbury, J Jour Biol Chem, 78, 47, 1928
2 Asken, J M Am Heart Jour, 22, 1, 1941
3 Atkinson, F R B, and Weber, F P Brit. Jour Dermat, 50, 267, 1938
4 Barber, H W Proc Roy Soc Med, 37, 73, 1944
5 Bauer, W, Marble, A, and Bennett, G A Am Jour Med Sci, 187, 237, 1931
6 Bevans, M Am Jour Path, 21, 25, 1945
7 [illegible], 130, 570, 1946
8 [illegible]
9 [illegible]
10 [illegible]

* Kindly supplied by Eli Lilly and Co.

The *synovial membrane* showed an outer layer of dense fibrous material and a layer adjacent to the joint space of greatly variable depth, composed of vascular, highly cellular connective tissue It was observed that *villous projections* branched from this tissue in some areas while in others, particularly where the synovia overlay tendons and ligaments, the layer was thinner and showed no villi

Abnormalities in the articular structures first appeared in the second decade and increased with advancing age The earliest changes occurred in the cartilage, which first became more opaque, yellowish and less elastic



FIG 220 —Anterior view of interior of knee joint of female, aged thirty-one years, average degeneration for decade Synovial membrane normal Note increased prominence of margins of femur, with irregularities of cartilage surface of patella and femoral groove

Its surface became slightly uneven and granular, with shallow linear furrows extending in all directions With increasing age, these became more [illegible] The surface [illegible] n strips or [illegible] ppearance.
Eventually (by the sixth decade) actual *ulcerations* of the cartilage were [illegible]

The overlying skin may become inflamed especially over pressure points such as knees and elbows, and the underlying calcific mass may suppurate The discharge may then contain calcium granules in gritty form, or a paste-like substance, and, occasionally solid white accumulations of calcium may be seen deep in the wound. These usually heal slowly without much scarring. They may be evacuated surgically. Their frequent occurrence near joints has led to the term 'calcium gout.' Calcium may also occur as 'sand' in the skin, or in plaques beneath the skin. The circumscribed masses are commonly found in the fingers and near the small joints of the hand.

These deposits cause little real disability except local pain and tenderness. They do not of themselves cause crippling, and may be overshadowed by the coexistent scleroderma.

The serum calcium and phosphorus values are generally normal, and there are no outstandingly abnormal laboratory findings except the *x*-ray picture. Here circumscribed, lobulated deposits are revealed in and under the skin and in the vicinity of small and large joints. They may vary in size from barely detectable granules to massive deposits with the apparent volume of a golf ball or a small orange. No deposits occur in the parenchymal organs (Figs. 210, 213 and 214.)

Interstitial calcinosis, known also as universal or diffuse calcinosis, presents quite a different picture. It commonly occurs in children or in young adults, and has been reported at the age of two years. An acute infection may precede it, and the first manifestation may be a scleredematous affection of the skin This is accompanied by fever, malaise, and diffuse muscle pain and weakness—much as in the clinical picture of dermatomyositis. Later, there is weight loss, emaciation and atrophy of muscles and disappearance of subcutaneous fat. The skin is thickened, adherent to the underlying structures, and may contain numerous nontender, firm, discrete nodules There is great stiffness of the muscles and ultimately limited motion of joints and deformity due to atrophy and fibrous contractures of the muscles. Splenomegaly has been reported Recovery has been reported, but untreated cases may die of the disease

[illegible] elevated, also the total protein. X-rays show [illegible] neous tissues and fascial

[illegible] of the interstitial connective tissue [illegible] The muscles are degenerated but [illegible] should be considered as primary calcinosis, or as a form of dermatomyositis with intercurrent calcification of the diseased tissues is difficult to judge.

Diagnosis.—The diagnosis depends on recognition of cutaneous and subcutaneous calcium deposits, and is usually confirmed by *x*-ray. Metastatic calcifications due to chronic renal failure, vitamin D intoxication, hyperparathyroidism, and other causes can be differentiated by the various features which respectively characterize these syndromes and by the fact that the calcium deposits occur in parenchymal tissues, whereas they are [illegible]

Where ulceration had occurred, the exposed subchondral bone had under-

development of marginal elevations it will be reported in detail Up to the age of fifteen the peripheral margin of the entire tibial epiphysis and that of nearly all the femoral epiphysis was found to be covered by a layer of



A

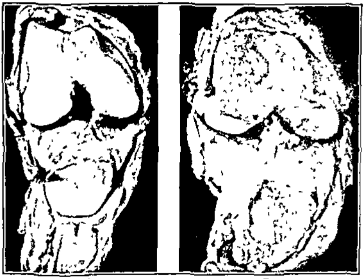


B

... of thirty-five ... A The ...rmal and most

hyaline cartilage nearly as thick as the articular cartilage itself. The fibers

same for vertical growth. At this stage of development, increased prominence was first observed at the junction of the synovial membrane and the periarticular margin



A B

FIG 222 —Opened knee joints from two females in sixth decade A Average degener-

With advancing age, the perichondrial margins became elevated and nodular, later overhanging Marginal "*lipping*" was observed as early as

abnormal *clusters of cartilage cells* appeared In later years, these changes increased in prominence to the point where deep vertical *clefts* and *pits*

Symptoms of Degenerative Joint Disease

Usually an elderly patient with few joints involved

Joints most frequently involved Terminal phalangeal joints of the fingers, lumbar vertebræ, knees, sacro-iliac, lower cervical vertebræ, hips and shoulders

Almost never involves wrists, elbows, knuckles or feet (except "bunion joint")

Usually *very little or no joint effusion*

Symptoms uncommon before forty years of age, except when due to trauma or artificial menopause [43]

Onset insidious in most cases

Patient usually overweight, hypersthenic type

Practically no systemic manifestations (fever, loss of weight, anemia, sub-

M

Pain usually is relieved by rest Stiffness after prolonged rest is relieved by "limbering-up" exercise

No significant muscular spasm, atrophy or flexion deformity

True bony ankylosis does not occur

Laboratory Tests

Sedimentation rate—usually *normal*

Complete *blood count*—usually *normal*

Agglutination reactions with hemolytic streptococci—usually *negative*, antistreptolysins—generally normal, antifibrinolysins—usually absent, usually no increased skin sensitivity to streptococci

Normal values for blood calcium, phosphorus, urea, proteins, albumin-

detail in the Chapter on Radiology [16, 32, 34, 35, 66] Roentgenologic hypertrophic changes may be seen in other forms of arthritis (gouty,[43] traumatic, gonorrheal, psoriatic or in some cases of rheumatoid arthritis[30, 43])

most often being, probably, environmental, less frequently perhaps, constitutional or inherited, but that the distribution between the two sexes is approximately equal if all manifestations of the disease are taken into account."

Joints Involved —*Primary degenerative joint disease* usually affects weight bearing joints (knees, hips, lumbar spine), and the shoulders, cervical spine and terminal interphalangeal joints of the fingers. Other joints

became evident. At times the clefts extended to the calcified layer which became partially separated from the bony epiphysis. In such areas chondrocytes appeared swollen or were in clusters, eventually, by the fifth decade, many disappeared. When the whole thickness of articular cartilage was involved, the subchondral bone marrow spaces were filled with cellular hyperemic fibrous tissue and osteoblasts
ance of
Lipping
chondral bone above the original cartilage-marrow border at the perichondrial margins. Such changes appeared in the fifth decade and were well-marked in the sixth and later decades.

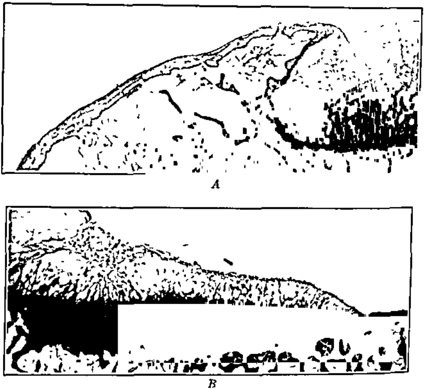


FIG 223 —Sections from the patellar surface of the femur of knee joint shown in *B* of previous figure (low power) *A* Marked elevation of the subchondral bone (lipping) is present at the perichondrial margin *B* Extensive degeneration of cartilage is apparent The ablation of this cartilage in the process of weight-bearing and motion has resulted in exposure of subchondral bone (eburnation)

The *synovial membrane* remained normal much longer than the other articular structures Most changes involved an increase in number and size of the *villi* with a gradual increase in the amount and density of the subsynovial tissue. The ends
evidence of degeneration. By
2 cm. in length) were given off
over fat pads and in areas of the joint lined by cellular areolar tissue

not infrequently involved include the first metatarsophalangeal, temporomandibular and proximal interphalangeal joints of the fingers Involvement of the latter, however, (in contrast to rheumatoid arthritis) is uncommon until after several of the distal interphalangeal joints are affected.

Although many other joints may show *pathological degenerative change, clinically* the disease only occasionally produces symptoms in them.

arthritis, does not occur and few patients present simultaneous symptoms in all of the joints mentioned above

Body Build.—Recent studies by Seltzer,[40,41] using anthropometric

ment and skeletal robusticity in the former, with strong laterality and

it is doubtful that these symptoms are related to the disease.

The cardinal *symptom* of this disease is *pain,* which occurs on use ("friction effect") and is relieved by rest. Such pain is usually aching in character and rarely is intense. Frequently, as with various other types of joint disease the pain is *intensified just before changes in the weather. Stiffness* after sitting ("articular jelling") and occasionally on arising is a common complaint. Such stiffness, however, is relatively short in duration and rarely persists for more than a few minutes; this is in contrast to the stiffness of patients with rheumatoid arthritis, in whom it may persist for several hours. Other subjective symptoms dependent upon sites involved will be mentioned under appropriate headings.

Objectively, joints may show localized *tenderness, crepitus, creaking* or *grating* and *enlargement.* When the joint is enlarged, the *swelling* usually feels hard Rarely, a slight increase *in synovial fluid* is present.

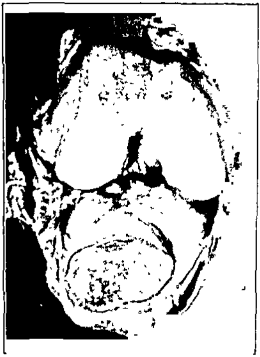
Laboratory Findings.—There are no specific laboratory abnormalities The sedimentation rate occasionally is slightly elevated (for unknown reasons) but is normal in most cases. Blood counts, urinalyses, blood chemical determinations, etc., are normal (See adjoining Summary) The basal metabolic rate is found to be lowered in some patients but by no means is this a consistent finding When effusions are present the synovial fluid shows normal cytological and chemical values except for inconstant elevation of the mucin content.[1]

Roentgenographic Appearance.—In some cases the roentgenographic appearance is normal Many gradations of abnormality may be noted in others The principal changes are those of *joint narrowing* (due to degeneration and disappearance of articular cartilage); *sharpening of articular margins and intra-articular structures* (*e. g.* tibial tubercles, etc.), *bony sclerosis* (*eburnation*); *osteophytes* and *marginal lipping,* and *bone cysts.*

...degenerative

eno-

nose



A



B

Patella shows advanced degrees of degenerative change

it. In evaluating such changes, one should also *x*-ray the opposite side when a joint of an extremity is examined The age of the patient and the normal physiological aging must ever be kept in mind. It should always be remembered that specific infectious arthritis and rheumatoid arthritis do not show roentgenographic abnormalities *early* in their course. When

as "*mixed arthritis*, although the degenerative changes may have no bear

gnosis of her-
diseases had
rthritis" was
ases, patients
were noted in
their roentgenograms. In neurotic patients, such information frequently

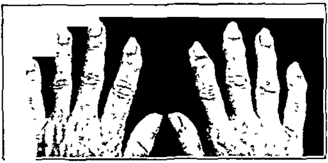


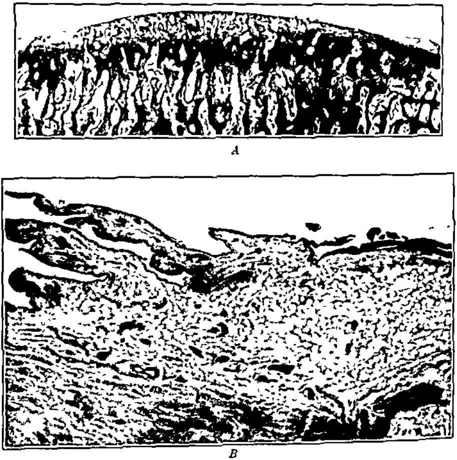
FIG 226 —Typical Heberden's nodes, the *signposts of degenerative joint disease* (osteo-arthritis) Note their typical *characteristic position* at the bases of the terminal phalanges, while the proximal phalangeal joints are not usually involved in this process

was followed by the development of pain which was rarely relieved by drugs or reassurance. It is common practise for some roentgenologists to draw arrows pointing to spurs Unfortunately, this practise tends to mislead the clinician and implant certain beliefs in patients' minds which may have *far-reaching adverse effects. This is true particularly in neurotic and* malingering patients, especially when compensation is a consideration

Specific Forms and Sites of Degenerative Joint Disease.—Heberden's Nodes —One of the most common manifestations of degenerative joint disease is the *Heberden's node.*[18] This is a cartilaginous and bony enlargement, *of the terminal interphalangeal* joint of the finger often characterized by flexion and deviation deformity. While Heberden's original description did not confine it to this joint, common usage has so designated it

Heberden's nodes may be single, particularly following trauma, but

Changes
the other
noted. V
tous char
mens was not proportional to the degree of arteriosclerosis in the peripheral or synovial vessels.



B of

is apparent Compare with Fig 173

the knee joints
degenerative
plantar apo-

neuroses.[40]

involved by this process but usually this does not happen until several of the distal joints are affected. Rarely, the metacarpophalangeal joints are involved. Heberden's nodes frequently are associated with degenerative joint disease elsewhere in the body.[44]

There are two forms of Heberden's nodes, the *traumatic* and the *idiopathic*. These are similar pathologically. Traumatic nodes may follow soon after a specific injury. They have been observed repeatedly in base-

swelling and *pain* over the involved joint. After several months, the acute

having been observed as early as the second decade. They are most *common in individuals who use their hands a great deal and women are affected* much more frequently than men (approximately 10:1).[44] Stecher found that *heredity* plays a large part in their origin, particularly in the female side of the family (mother, daughter, sister, etc.). He found that it was *twice* as common in *mothers* and *three times* as common in *sisters* of women affected as the normal expectancy in the general population.[45]

Clinically, these enlargements usually are a gradual development, often *painless*, which may progress unnoticed for months or years. In other cases, they appear rather rapidly, with *redness, swelling, tenderness,* and *aching*, particularly after use. Individuals afflicted by them frequently complain of *numbness* and *tingling* of the finger tips, often "*clumsiness*" on use. The *swelling* may be soft and fluctuant or may be quite hard. The joint itself may show sidewise *displacement* or *flexion* of the terminal phalanx. At times, the original swelling is complicated by the appearance of *small, gelatinous synovial cysts.* These appear generally on the dorsal aspects of the *terminal interphalangeal joint or just proximal to it.* They often are attached to tendon sheaths and resemble ganglia, they may recede spontaneously or may persist indefinitely. At times they precede the appearance of Heberden's nodes,[46] sometimes appear in individuals without them.

Roentgenograms reveal *enlargement* of the *ends* of the *bones*, with *distortion of joint space* and *irregularity* of the *joint surface*. Large *spurs* develop from the attachments of the flexor and the extensor tendons to the terminal phalanx. Although the enlargement usually feels quite hard, one is surprised to find that in many cases the *x*-ray shows only minimal bony spurs

bably are

ner joints

Symptoms begin usually in the fourth or fifth decades. ... the disease

from obesity; poor mechanics due to pronated feet, ...

and traumatic hemorrhage. Degenerative changes also are seen following

and 1914,[6] Bauer, Ropes and Waine,[9] 1940) it has been demonstrated that lacunar resorption occurs frequently in traumatized joints.

The cause of *marginal proliferations* (spurs, *osteophytes*, lipping, exostoses) also still is under speculation. It is possible that no single mechanism is operative in these changes but that advance of subchondral marrow, periosteal bone formation, endochondral ossification of cartilaginous matrix, compression of the devitalized articular covering, and certain definite structural and functional characteristics of the border zone between articular cartilage and synovial lining tissue all play a part at times. One must keep in mind, however, that *marginal proliferation is not an early sign of degenerative joint disease*

One other point of importance should be mentioned here. The extent of

surface becomes eroded and eventually worn down Such erosion proceeds in an irregular fashion so that the surface is uneven and the layer of cartilage varies in thickness Ultimately, portions of the cartilage may be worn

Such ex-

he cellular components of the uppermost layer appear more numerous, forming a dense layer, their long axes arranged parallel to the joint lumen. The immediately subjacent zones contain fewer cells The intercellular substance is swollen, the individual cells are voluminous and heaped in clusters

only in those portions of bone from which the hyaline articular covering was entirely missing Secondary degenerative changes may occur in these areas and produce so-called *"detritus cysts,"* which may be macroscopic in size and

and size of villi The villi may contain cartilage or may be partially ossified At times such villi and even portions of the bone or cartilage break off to form *loose bodies* in the joint.

Degenerative changes, comparable to the above, occur also in tendons, ligaments and aponeuroses

burnt-out specific infectious and rheumatoid arthritis, tabes dorsalis and syringomyelia.

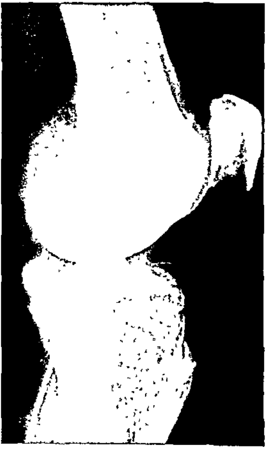


FIG 227 —Early stage of degenerative joint disease of knee with slight bone production Little narrowing of joint space (Doub, courtesy of Radiology)

Objectively, the joint shows very little Findings may include *tenderness* over the lower margin of the patella, *pain on passive motion* of the patella over the femur and tenderness over the joint line or medial aspect. *Crepitus*

While the changes described above are characteristic of *peripheral joint* involvement, degenerative joint disease of the spine, so-called "*osteoarthritic spondylitis*" or "*spondylosis*" produces similar changes Because of mechanical differences, however, as well as the fact that the intervertebral discs are not true joints, certain differences are noted

Collins[19] points out that "osteoarthritis" is a disease of *synovial (diarthrodial)* joints, with a pathological picture as mentioned above. Since the intervertebral joints do not contain synovia, true osteoarthritis of the spine can occur only in the apophysial and costovertebral joints Actually, he found such changes quite frequently in apophysial but only rarely in costovertebral joints

Thus, the disease may exist without marginal lipping of the vertebral bodies. The latter feature characterizes spinal *osteophytosis* and results from an entirely different pathogenic mechanism, namely a disorder of the intervertebral disk.

Osteophytes cannot form so long as the nucleus of the intervening disk is intact and the vertebral bodies are normally separated. Collapse of tht disk, however, through degeneration or prolapse of the nucleus, leads te slight forward tilting of the vertebræ and the forward extrusion of whoa plastic substance remains It is on either side of this extruded disk substance that osteophytes subsequently develop, apparently because of stram on the vertebral ligaments which results in periosteal irritation and proliferation of bone. Usually, osteophytes occur in an anterolateral position (usually the right side) just to one side of the anterior common ligament, whose strong attachments are sufficient to prevent the extrusion of the disk in the mid-line anteriorly.

Disk collapse and osteophytosis of the vertebral bodies soon leads to immobilization of the affected spinal segments, a condition which actually should prevent degenerative changes in the apophysial hoints (since they are put at rest) The disks, themselves, become dehydrated, fibrillated, fissured, thinned and less elastic. The end picture is one of cartilage destruction with narrowing of intervertebral joint spaces, marginal bony spurs and sclerosis of juxta-articular bone.

As with the degenerative joint changes in peripheral joints, the process in the vertebræ and disks increases with age Schmorl[56] studied ten thousand spines during postmortem examinations and found that osteophytes were rare during the first twenty years of life but increased in frequency with age following that period. They were present in 11 per cent of subjects in the third decade, 36 per cent of those in the fourth, 78 per cent in the fifth and 93 per cent of those in the sixth.

Certain portions of the spine are affected more commonly than others. Thus, Shore,[57] in a study of 126 vertebral columns with "osteoarthritis," found no involvement between the first and second cervical vertebræ and only very infrequent changes between the eight cervical and fourth thoracic vertebra. The most commonly involved areas were (1) between the third and sixth cervical vertebræ, (2) the fourth and tenth thoracic vertebræ and (3) between the third and fifth lumbar vertebræ.

Horwitz[34] analyzed the changes in cervical spines of 50 adult males. Seventy-six per cent showed varying degrees of intervertebral disc degeneration. Changes were most common in the lower cervical segments, involving the discs between the fifth and sixth vertebræ in 76 per cent, between the fourth and fifth in 56 per cent and between the sixth and seventh in 36 per

cytological and chemical findings.[1] Muscle spasm and atrophy about the knee are uncommon unless disuse has been prominent.

Degenerative Joint Disease of the Spine.—On the basis of roentgenographic observation of articular lipping and spur formation (osteophytes) on the bodies of spinal vertebrae, the spine has been cited as the most common site of degenerative joint disease in men (and second to Heberden's nodes in women).[19] It is now recognized, however, that *such findings may be entirely*



FIG. 228 —Later stage of degenerative joint disease of knee with more extensive bone production and joint narrowing (absorption of cartilage) (Doub, courtesy of Radiology.)

asymptomatic and of no particular clinical significance. Indeed, as mentioned in the preceding chapter, such changes are characteristic of "spinal osteophytosis," while true degenerative joint disease (of diarthrodial joints) is relatively uncommon. Thus, Cocchi[6] found that of 81 patients radiographed because of other symptoms (tumors of the neck, goiter, *etc.*), and *without a history suggestive of degenerative joint disease,* 50

cent. Marginal exostoses were present in 70 per cent, being most marked on the anterolateral portions of the vetebral bodies, particularly of the lower four cervical vertebræ. In 40 per cent, encroachments of intervertebral foramina were present. These were due to posterior exostoses on the vertebral bodies and/or to anterior proliferations at the margins of the articular facets

Although exostoses at times may be of sufficient length to coalesce, real *ankylosis does not occur* Degenerative processes may be found in the capsules of the small posterior intervertebral (*apophyseal*) joints and in the paravertebral ligaments, where spotty calcific deposits sometimes can be demonstrated.

The cause of degenerative changes in articular and periarticular structures is unknown Bowie and his associates found small but well-defined respiratory activity ir [illegible] this activity with incr [illegible] respiratory power of [illegible]

Pathogenesis of Degenerative Joint Disease

1 *Degenerative changes* occur *in the hyaline articular cartilage* as a result [illegible]

[illegible]

menisci, synovialis and in further damage to the hyaline cartilage

4 The weight-bearing joints (especially the knees and hips) show a numerical and quantitative predominance of involvement with the [illegible]

[illegible]

signs of degeneration

7 In those patients with marked cartilaginous lesions, *subchondral condensation of bone* appears

8 With the decreased substance and elasticity of the degenerating [illegible]

[illegible]

attritional forces of joint function

10 *Effusions into joints* are *rarely present* (and then are small) in degenerative joint disease The delicate intima of the synovialis may be traumatized mechanically by the gross deformity of the articular surfaces, resulting in a few instances of limited histologic evidence of inflammation

11 Secondarily, *villi* may appear adjacent to the articular margins permanent folds which have undergone hypertrophy probably as the result of mechanical irritation or pinching between the irregular surfaces of the joints.

per cent showed osteo-arthritic changes of various degree. Breck, Hilliman
of the lumbar spine
heavy work, that
cent *mild* changes.
wing of the lumbo-
sacral space. In recent years, many individuals showing such changes have been found to have *herniated nucleus pulposus.* After removal of these

arthritis") of the spine solely on the basis of such findings.

That this disease does exist in the spine is undisputed, but it is necessary

other, which may be followed by stiffness of the spine and decreased motion when fibrosis of the disk has occurred [29]

which pains radiate into the arms or legs Often, pain is produced in such cases by compression of the nerve roots resulting from narrowing of the intervertebral foramina through which these pass, such narrowing commonly is due to thinning of the intervertebral disks, particularly in the cervical spine. Here, the foramina are small in comparison to the size of the nerves which emerge from them and the nerves are easily encroached upon [23] It is possible, however, that the radicular pain might be due to *intraspinal prolapse of nucleus pulposus,* since this lesion also is followed, in some cases,

close approximation of the vertebral bodies from thinning of the intervertebral disks As a result of these two processes, the nerve trunk may be im-

changes) (See also under Chapter on Low Back Pain)

Cervical Spine.—Because of the varied and numerous ramifications and interconnections of the cervical nerves, pain in many regions may result from disease in the cervical vertebrae with involvement of the foramina (Fig 229) Thus, they may cause occipital headache, earache, unilateral sore throat, neuritic pains in the arms and fingers, etc In some cases, thoracic pains simulating *angina pectoris* may occur when the neck is turned in certain directions [31]

generative joint disease in aging man
tion of costal cartilage suggests
chondroitin sulfate.[43,61] It is pos

cartilage. It is known that articular cartilage has no blood supply, a low respiratory rate and a very limited faculty of autogenous repair. *Attempts* at healing result usually in an immature form of fibrocartilage rather than hyaline cartilage.
changes in cartila
pressure, weight ar
in this chapter, under Etiology.

Marginal proliferations have been attributed to various causes and it is probable that they have several modes of genesis Many workers have concluded that stimulation of
a great part in this productio

(1934) attributed exostoses to projection of bone and cartilage over the
or
fo
calcification of cartilaginous matrix and outgrowth of subchondral bone Bennett, Waine and Bauer,[14] acceding to most of these explanations, believed that *no single mechanism was operative in all cases* and suggested that one type of marginal proliferation was due to certain definite structural and functional characteristics of the border zone between articular cartilage and synovial lining tissue. They pointed out that in some instances marginal ridging was discernible before any definite subchondral reaction or flattening of articular surface had occurred and therefore should not be considered secondary to these developments.

BIBLIOGRAPHY

1. Amprino, R, and Bairati, A: Ztschr. f. Zellforsch u mikr. Anat, 20, 143, 1933
2 ———: *Ibid*, 20, 489, 1934.
3. ——— *Ibid*, 21, 448, 1934.
4. Assmann, H: Fortschr. a d. Geb d. Roentgenstrab, 33, 901, 1925
5 Axhausen, G: Arch f. Klin Chir., 99, 519, 1912
6. ——— *Ibid*, 104, 301, 1914
7.
8.
9.
10.
11 Bell, B Remarks on Interstitial Absorption of the Neck of the Thigh-bone, Edinburgh, Maclachlan and Stewart, 1824
12. Beneke, R. Zur Lehre von der Spondylitis deformans, Versamml d deutsche Naturf. u Aerzte. Braunschweig, 1897.
13 Bennett, G A, and Bauer, W.: Jour. Clin. Invest, 13, 700, 1934.
14. Bennett, G A, Waine, H, and Bauer, W. Changes in the Knee Joint at Various Ages, New York, The Commonwealth Fund, 1942
15 Bowie, M. A., Rosenthal, O., and Wagoner, G: Ann. Rheum. Dis, 2, 216, 1941.

in only 3 patients. Most of these patients showed some sclerosis of th articular facets.

tis, there was no noticeable narrowing of the intervertebral foramina o thinning of the intervertebral disks.

Degenerative joint disease of the cervical spine must be differentiate from cervical ribs, the scalenus anticus syndrome, herniated nucleus pu posus, fractures and dislocations of cervical vertebrae, traumatic strains

Each patient must be studied carefully before the diagnosis is established Since the roentgenographic findings described above frequently ar *asymptomatic and may appear as a physiological manifestation of aging* one must not accept these findings without careful appraisal of the symp toms and signs. *In the absence of actual mechanical impingement or com pression of nerve fibers by narrowed intervertebral disks, or roentgenographi changes in the apophyseal joints one must proceed cautiously before attributin symptoms to degenerative joint disease.* Even when these changes are present each case must be evaluated by the *composite picture of all factors.*

Thoracic Spine.—*Degenerative joint changes in this area may produc* localized aching or radiating pain (radiculitis). Localized aching, however is probably due more to chronic muscle strain than to actual joint disease Such strain may result from poor posture, old osteochondritis dorsalis juvenilis, and various occupations. In some women, the strain of supporting very large, pendulous breasts may lead to undue stress on the thoracic spine.

Several writers have brought out the fact that dorsal root pain due to

It may be paroxysmal and be noted simultaneously in one or both arms. Willius[51] states that such pain may become more pronounced when the patient is reclining. Smith and Kountz observed 15 patients who had dorsal root pain *simulating angina.*[42] They remarked about the difficulty in recognizing the cause of such pain. In patients with dorsal root pain, the pain may occur on exertion, may be of short duration and may be

greater than in angina pectoris Davis[7] recently reported 10 cases chest pain simulating coronary artery disease. He stated that attacks occurred (1) at night; (2) after such movements as bending, turning, etc., and (3) after such acts as coughing, sneezing, straining at stool or prolonged sitting. Most important diagnostically was the *reproduction of such attacks by pressure over the dorsal vertebrae* and the *cure* by proper management.

Oppenheimer believed that segmental neuritis developed from crushing of nerve roots due to thinning of the intervertebral disks and collapse of the intervertebral foramina.[34] Others have also reported that osteophytes

24 FISCHER, A G T.. Internal Derangement of the Knee Joint, New York, The

3, 156, 1035
32 Francis Adams,
33 Skeletal Remains,
34
35
36 KAHLSTROM, S C, BURTON, C. C, and PHEMISTER, D B · Surg, Gynec, and Obst, 68, 129, 1939
37 KEY, J A · Jour Lab and Clin Med, 15, 1145, 1930
38 ——— · Am Med, 36, 610, 1940
39. KLING, D H Am Jour. Med Sci, 197, 358, 1939
40 LEDDERHOSE, G Die Arthritis deformans als Allgemeinerkrankung, Strassburg, K J Trubner, 1915
41 LINDE, F · Munchen med Wchnschr, 79, 2009, 1932

ed,
53, 325, 1934
48 ——— Arch of Path, 17, 516, 1934
49 POMMER, G. Mikroskopische Befunde bei Arthritis deformans Denkschr d k Akad d Wissenoch, Wien, Holder, 89, 1913
50 ——— Virchows Arch f path Anat, 219, 261, 1915
51 ——— · Ueber die Beziehungen der Arthritis deformans zu den Gewerbekrankheiten Das osterreich Sanitatswesen, Wien, Holder, 1918
52 ——— Virchows Arch f path Anat, 263, 434, 1927.
53 POTTER, D G E Harrowgate Spa Med Jour, 1, 10, 1938
54 RAWLS, W B, RESSA, A A, GRUSKIN, B, and GORDON, A S Ann Int Med, 11, 1401, 1938
55
56
57
58
59
60
61
62
63
64
65.

frequently occur about the transverse processes, spines and foramina, with *resulting irritation of nerve roots.* However, *Key feels that,* in most instances at least, there is insufficient osteophyte formation in the vicinity of nerve roots to account for the dorsal root irritation [24]

In Smith and Kountz's series, *postural defects* were evident in all of the patients.[42] They attempted to determine (by a study of the spinal nerves



Fig 230 —Marked hypertrophic changes in the lumbar spine

in situ) whether chronic deformities of the spine and thoracic cage might exert traction on the nerves with a resulting stimulation, particularly of the

CHAPTER 39

SYMPTOMS AND LABORATORY FINDINGS OF DEGENERATIVE JOINT DISEASE: (OSTEO-ARTHRITIS)

By Nathan R. Abrams, M.D.

Incidence.—The preceding chapter makes it evident that from a pathological standpoint, degenerative joint disease is present universally in individuals beyond the second decade of life. Roentgenographically, *manifestations of this disease do not appear as a rule until the fourth or fifth* decade, but after the age of fifty characteristic changes are present almost universally. Despite these facts and the fact that individuals may complain of symptoms in the absence of x-ray abnormalities, only about 5 per cent of individuals past fifty have clinical symptoms.[19]

For these reasons one cannot help but wonder if too much attention has not been paid to morphological changes. Various features have been offered in explanation of the symptoms of this disease. These include elevation of

unsettled.

Climate.—Clinically, this disease is essentially one of the north temperate zone. However, many World War II soldiers stationed in South *Pacific tropical islands first developed symptoms while in these* areas; others who had the disease prior to military service experienced aggravation of their symptoms in the tropics.[19] It has been stated that less "rheumatic disease" is present in Eskimos and Lapps than in individuals residing in the temperate zone.[21] In general, patients with degenerative joint disease are *affected less by changes in the weather than are patients* with rheumatoid arthritis. However, they frequently complain of more pain while in damp areas and cold.

Age.—In the absence of specific trauma or previous infectious arthritis, degenerative joint disease is an affliction of middle or old age. However, *experiences in an Army Arthritis Center* revealed the fact that it was common during the fourth decade of life.[19] In women, it often makes its appearance at the time of or soon after the menopause.

Sex.—Goldthwait, Painter and Osgood[16] stated that men are *more frequently* affected than women and that the large joints are involved more often in the former. Swaim[17] believed males suffered from the disease ... as often as females. Nichols and Richardson[18] found that women

vere included. Dawson[5] and
neral sex distribution about
es and cervical spine show a
r spine and hips. Bennett,

especially at their angulations through the spinal foramina and at their attachments to the cord. They concluded that movement imposed on the nerves under such tension might give rise to irritation of the nerve fibers with the production of referred pain.

Lumbar Spine.—Here, degenerative joint changes commonly are a normal accompaniment of age, particularly in those doing heavy physical labor Such changes become more marked in the presence of obesity or altered body mechanics due to poor posture or faulty mechanics in the feet and legs. They result also from old strains and injuries.

Because of the frequent roentgenographic findings of marginal lipping and narrowed intervertebral disks in the lumbar spine, probably more errors are made in diagnosing low back conditions than of any other single malady. In evaluating such changes it must be realized that they may represent secondary results of other conditions rather than the primary cause of symptoms.

Degenerative joint disease of the lumbar spine frequently must be diagnosed by exclusion. As in other sites of the body the symptoms characteristically are those of *aching* on use with relief by rest, and muscular *stiffness* Physical examination may reveal paravertebral tenderness and limitation of motion. The roentgenogram reveals changes as described above Sclerosis, osteophytes and narrowing of the articular facets also are found on *x*-ray. Hypertrophic lesions may encroach upon emerging spinal nerves and may simulate symptomatically herniated nucleus pulposus.

Degenerative changes in the *lumbosacral joint* probably cause more symptoms than in any other site in the low back. Whether such symptoms are due to degenerative disease of this joint or to herniations of this disk ... is open to question ...

... must be differentiated ... disks, infectious bone disease (tuberculosis, undulant fever, typhoid fever, etc.), rheumatoid spondylitis, osteoporotic conditions, Paget's Disease, spondylolisthesis, traumatic and structural strains, and "fibrositic" conditions Various ... conditions such as prostatitis, various female pelvic conditions, ... ulcer also must be excluded. (See

Sacro-Iliac Joints.—Roentgenographic changes of marginal lipping, sclerosis of the articular margins and narrowing of the joint spaces have been found with increasing age.[43, 52] Ankylosis also has been described but probably such a finding represents an old burned-out infectious or rheumatoid arthritis.

Normally, strong ligamentous attachments prevent any measurable degree of motion in the sacro-iliac joints Symptoms may appear when *relaxation of these ligaments permits motion of the joints* and stretching of surrounding structures. Such symptoms include *low backache* and *pain*, aggravated by standing and by movements of the leg which cause motion *in the sacro-iliac joint or by rising from a recumbent position*.[22] Frequently, the pain is referred down the legs. Localized *muscular spasm* often is ... the concavity of the resulting lateral spinal curvature toward ... in the sacro-iliac joint on straight ... limited.

These symptoms, combined with the above mentioned roentgenographic findings, have been accepted generally as evidence of degenerative joint

disease of the sacro-iliac joints. In recent years, however, it has been

of the sacro-iliac joints is quite rare.



FIG 231 —*Degenerative hip joint disease* Note almost complete loss of articular cartilage, flattening of the femoral head and small cystic areas in the head and neck of the femur Subchondral bone is condensed

Degenerative Hip Joint Disease· (Malum Coxae Senilis, Morbus Coxae Senilis) —This condition has been referred to as degenerative arthritis,

etc.) may produce hyperemia and relieve pain temporarily. Practically never are narcotics required; when such drugs are necessary to relieve joint pain one should suspect malignancy.[40]

Phenylbutazone (Butazolidin).—This drug has recently been recommended for relief of pain in osteoarthritis by Kuzell and others.[24] They treated 11 cases in whom there was a mixture of osteoarthritis and rheumatoid arthritis and obtained marked relief of symptoms in all but 1 case. Hollander[21] has treated 27 osteoarthritic patients with the drug, in doses from 600 mg. to 800 mg. per day in divided doses, and found that less than half his patients obtained greater relief from phenylbutazone than from

oo toxic, and
use in most

Cortisone and Adrenocorticotropic Hormone (ACTH).—Hench and his associates[19] noted relief of joint pain from cortisone in a patient with leukemia, who had incidental "primary osteoarthritis." Boots and Ragan[2] reported beneficial effects from this drug in patients suffering from degenerative joint disease of the hips (malum coxæ senilis). Thorn and his associates[49] observed benefit in one case of osteoarthritis treated with ACTH. Improvement from the use of this hormone was also reported in a case of

et al.[74] In one case of painful Heberden nodes this author noted no improvement with cortisone.[40]

It is to be expected that because of the analgesic effects of these drugs, temporary alleviation of pain might result in occasional cases during the administration of either of these substances. However, because of the equivocal results from such therapy, the relatively innocuous nature of most forms of this disease, the expense involved and the possible deleterious systemic side effects from long continued administration, treatment with either cortisone or ACTH is not recommended.

Intra-Articular Hydrocortisone.—Hollander, Brown, Jessar, and Brown[20] have reported successful relief of symptoms in osteoarthritis of peripheral joints in a large series of patients by injection of a suspension of hydrocortisone-acetate directly into the joint space. Repeated injections have

symptoms in this disorder. Strain, fatigue and instability follow when the weight thrust is changed and the function of the muscles about the hip is impaired, with the forward inclination of the pelvis and flexion of the hip

...the hip, groin or

Pain is present

...ally in the groin

or along the inner aspect of the thigh; at times it may be referred to the buttock or sciatic region. The degree of pain varies widely and does not always depend upon the amou...

creases following sitting or rest

companies initial motion from

provide some relief, *violent exertion* may aggravate the condition. Severe backache may result from the compensatory lordosis which follows fixed flexion. Abduction and rotation become markedly limited. In advanced

density in the postero-superior border of the acetabulum and the antero-superior surface of the head of the femur. (At this stage, motion of the hip is limited although subjective symptoms may not appear for several years). Later roentgen changes include osseous proliferation around the posterior ... head and neck of the

...ed or

the weight-bearing portions of the femoral head and acetabulum. Inasmuch as *only the exostoses at the superior and inferior portions of the joint are visualized*, the abundance of these outgrowths is not appreciated usually in the roentgen study. For this reason, the roentgenologist must be especially careful in the wording of his report.

...the anterior and superior portions of the joint. The roentgenologi...

of subarticular bone and small areas of irregularity of the...

Flattening of the head of the femur may result in a *mushroom* appearance over the neck of the bone. The head of the femur may be displaced upward and outward and actual subluxation may occur.

joints. It is only helpful in about half of the patients with malum coxæ senilis, but perhaps is worth a trial before orthopedic surgery is attempted
'nal joints since these are anatomically
A full discussion of the method for
apter 18. Hydrocortisone injections
into the osteoarthritic joint are an *adjunct, and do not supersede the preceding time-tested measures.*

Iodides.—These have been employed for many years on an empiric basis. Usually 0 3 to 0.6 cc. of a saturated watery solution of potassium iodide has been used 3 times a day. The drug must be discontinued or



Fig. 232 —Close-up of Heberden's nodes affecting index and middle fingers

Harmon has described a *secondary form of degenerative hip joint disease* occurring in individuals two or three decades younger. In his series of 94 patients, the mean age of the secondary cases was forty-five years, and of the primary cases, sixty-four years. There were no essential differences pathologically between the hip joints of the primary or secondary group.[17]

Various authors have enumerated the conditions that may predispose to degenerative hip joint disease Plewes studied a series of 242 cases and found preexisting hip joint disease in over half of the cases (as trauma, adolescent slipped epiphysis, coxa plana and the Otto pelvis-protrusio acetabuli).[25] Wiberg also discussed the occurrence of degenerative hip

t

Others have detailed the changes occurring in bone and joint cartilage following *avascular necrosis*[25 26 27] Harmon felt that avascular necrosis,

changes in the femoral head which follow traumatic dislocation of the hip[26] Similar changes have been shown to occur (experimentally) in the hip joint after denervation

Harmon thought that in some instances the subchondral osseous infarc-

chondro-osseous overgrowths (osteophytes)

location of the hip, congenitally inadequate acetabulum, congenital anomalies in the upper part of the femur, injury, fracture of the pelvis into the acetabulum, fracture of the neck of the femur followed by avascular necrosis of the femoral head (with and without the use of foreign body fixation),

episode (sometimes resulting in fracture) or minor repeated trauma of occupational or everyday activity, in addition to a hereditary predisposition to cartilage injury and/or degeneration. About one-third of his patients were obese, obesity could increase the strain upon the joints of the lower extremity

When *damage to cartilage* has been produced, a vicious cycle of *added trauma* and *destruction of cartilage* occurs until the *underlying bony cortex*

decreased if pustular eruptions, coryza, gastro-intestinal upsets or other signs of iodism develop. Few rheumatologists use this drug today.[40]

Vitamins.—Vitamin D in massive dosage has been recommended for degenerative joint disease as well as for rheumatoid arthritis.[10 25 43 45] Its use is empirical. Dosages of 50,000 to 500,000 units per day have been

reactions include nausea, vomiting, pruritis, headache, polyuria, polydipsea, mental confusion, vertigo, renal calculi, subcutaneous calcifications,

oth

of

suc

an

me

Heberden's nodes.

Other Drugs and Measures.—*Roentgen therapy* has been recommended by some workers[9 11,22 23 26 38 53] but is generally not utilized by most experienced rheumatologists Kuhns and Morrison advocate a total dosage of

d their results in

marked improve-

cent, slight im-

provement in

gators recomm

3 to 12 treatm

without much (if any) effect on the disease itself.[9] Scott, however, believes it reduces congestion and leads to absorption of fibrous tissue [37 49] Stamm recommends roentgen-therapy for deep, boring pain, presumably caused by bone sclerosis [45] In this author's experience roentgen therapy has been of little value in relieving pain, of no value in altering the disease process

jected under sterile precautions at the site of maximum tenderness. Such

but usually are harmless

may be *bared*, with resulting changes *in the subchondral bone.* Harmon stated that rheumatoid or purulent arthritis could produce damage to the articular cartilage; this was, in some instances the forerunner of later degenerative hip joint disease. He commonly found wedge-shaped sequestra produced in the femoral head by avascular necrosis (one-third of the cases) and suggested that this had probably initiated the joint damage in those cases.[17] No bacteria *were cultured from any of the affected hip joints.* There seemed to be no important endocrine abnormality, except the obesity (which was postmenopausal in some cases). Juxta-articular adiposis [illegible]

head may resemble the pitted surface of a golf ball, with similar changes being seen on the acetabular side.[17] The typical *exostoses* are evident immediately.

When cartilage and bone are removed, as in an arthroplasty of the hip, the amount and extent of anemic, avascular bone in the femoral head may be marked. This avascular bone is often wedge-shaped or conical with its base upward. The weight-bearing portion of the femoral head and the acetabulum show the most pronounced gross and microscopic changes

Harmon has described loose osteochondral bodies, thickened joint capsule, free joint fluid, and hypertrophic reddish-blue synovia in these joints It is probable that in the primary degenerative hip joint disease, very little *fluid will be noted. In his patients, the fluid was clear or slightly cloudy,* and straw-colored, and thick. When the surgeon dislocates these hips at operation, he is struck by the *increased size of the osseous portion of the femoral head,* which occurs *at the expense of the cartilage over the femoral head and in the acetabulum.*

Grossly, the first changes noted in these joints are a change in color and increase in translucency of the articular cartilage. Fibrillation of the carti- [illegible]

areas of cartilaginous degeneration. *Sclerosis of subchondral bone occurs* in some areas At the margins of the joint, proliferated bone pushes cartilage *before it, leading to the formation of chondroosteophytes.*

These *exostoses* probably are responsible for the marked limitation of [illegible] rotation and flexion) which is so common in the later [illegible] Unfortunately, the exostoses are [illegible] roentgenograms as they do not have a well-developed osseous cortex.

A large exostosis is usually found on the superior and anterior portion of the femoral neck. In some cases, this exostosis of the femoral neck may be very large and may completely bridge the depression between the head of [illegible] with

Another large osteophyte is often found on the inferior and [illegible] of the femoral head, as the adduction deformity increases, *this osteophyte*

Patients sensitive to procaine should be given some other local anesthetic such as intracaine. Patients should be warned that they may have increased pain several hours after the injection, when the effect of the procaine has worn off; such increase in pain probably is due in part to an increase in blood supply to the tissues infiltrated.

Recently, the use of *intravenous procaine* in 0.1 per cent solution has been recommended [13,14] In this author's experience in 20 cases[60] and Steinbrocker's in 32 cases,[35] such therapy has been almost worthless. In practically all cases, the relief from pain has lasted for only a few hours, and in no case more than a few days.

Injections or *iontophoresis* with *histamine* or *mecholyl* (acetyl-beta-methylcholine) have been advised for reducing local discomfort. Their effects, however, apparently are those of physical therapy and they have no advantages over many simpler forms of treatment.

Intra-articular injections of lactic acid have been recommended to stimulate natural local tissue reactions and evoke processes of repair in traumatized joints.[25 51 52] *Such therapy, however, has not been accepted by most* workers.

Removal of focal infection, gold therapy, sulfur, chaulmoogra oil, bee venom, fever therapy and sympathectomy have no place in the treatment *of degenerative joint disease. A few workers continue to advocate vaccines* but the overwhelming r [illegible]
staunch advocates of va [illegible]
reporting on the use of [illegible]
cent of 64 patients by [illegible]
suggesting that any improvement from the use of vaccines in this disease was psychologic.

TREATMENT OF SPECIFIC LESIONS OF DEGENERATIVE JOINT DISEASE

[illegible]

should be reassured that they do not have crippling arthritis; they should be advised to accept their appearance and not waste money on worthless treatments When pain is present, physical therapy in the form of paraffin baths, hot soaks or contrast baths usually will relieve it. The avoidance of *trauma not only reduces pain, but probably retards the progress.* As mentioned above, estrogenic therapy, in the author's opinion, sometimes relieves pain and numbness However, *Heberden's nodes ultimately become painless regardless of therapy* and often cause no pain at any time.

Cervical Spine Lesions.—*These lesions frequently* benefit from long rest periods, particularly while lying on the back *without a pillow*, combined with physical therapy in the form of diathermy, massage and graded exercises. Often, manual traction or suspension with gentle rotation aids *in overcoming muscle spasm and possibly* pressure on the cervical nerves from degenerative changes in the intervertebral foramina. McFarland and Krusen have employed the modified Sayre head sling after a thirty minute

becomes larger and more irregular. It also may articulate with a projection around the acetabulum and prevent flexion, rotation and adduction.

In addition to the above mentioned osteophytes, one may see a rim of osteophytes around the acetabulum projecting anteriorly and posteriorly,

and inferior portions of the joint, the remaining two-thirds of the head of the femur and margin of the acetabulum (the anterior and posterior portions) being covered by other shadows [17]

The subchondral bone becomes exposed as the cartilage degenerates The osseous articular cortex becomes tremendously thickened (*eburnated*)

trauma to adjacent nerves resulting in referred pain around the chest or abdomen [18] The 24 costovertebral joints are formed by the junction of the heads of the ribs with the side of the spine In addition to these, there are 20 costotransverse joints which are formed by the articulation of the angles of the ribs with the outer parts of the transverse processes of the upper 10 dorsal vertebrae

Each of these 44 is a true joint, all are involved in the mechanics of the thorax and may be injured or diseased. *The rib joints differ from other joints of the body in that they never rest during life* unless they are ankylosed as a result of disease The range of motion of the rib joints is such that there is a difference in chest circumference of 3 to 5 inches between full inspiration and expiration in normal individuals

At the level of each costovertebral joint, the intercostal nerve leaves the spine and passes along the rib Irritation of these intercostal nerves at the costovertebral joints results in many cases of pain referred to the chest wall or around the body at different levels The circulatory disturbances often seen in connection with irritation of these joints may be due to the fact that the ganglia of the sympathetic nervous system lie adjacent to the costovertebral joints

sternal cartilage, having no costotransverse articulation to limit motion of the main joint, they may sag and move to such an extent that a greater strain is placed upon the rib joint with a greater possibility of irritation of

costal neuralgia)

The best position for rest of the rib joints is midway between extreme expiration and inspiration In the normal chest, the average downward

procedure is carried out twice a day for a week to ten days, after this the patient is instructed in the use of the sling at home.

Other treatment includes *analgesics, orthopedic appliances* (Thomas collars, etc) and, at times, *local procaine injections* The latter often results in temporary relief, but must be followed by other measures

Thoracic Spine.—Treatment of symptoms resulting from disease in this area should include *rest, physical therapy* and the *correction or control of factors causing additional strain* (obesity, kyphosis, scoliosis, occupational strain, etc) Abnormally large, pendulous breasts should be supported properly to relieve some of the strain posteriorly Pendulous abdomens also should be supported

Davis, in describing radicular pain simulating coronary artery disease, noted relief of symptoms after postural correction, bed boards, exercises and particularly manipulation and traction of the cervicodorsal spine [7]

Lumbar Spine.—As remarked above, it is most important to rule out other conditions before accepting the diagnosis of degenerative joint disease of the lumbar spine Paupurt stated that when a worker over forty years old with symptomless, perhaps unsuspected, "hypertrophic spondylitis" falls down, the cost to his employer is likely to be about $3000 00.[25]

Degenerative changes in the lumbar spine may be associated with altered mechanics of weight bearing, abdominal adiposity and loss of tone of the back muscles Treatment is similar to that described for lesions of the thoracic spine Curtailment of activities, such as golf and other sports, usually is indicated Stout patients with abdominal sag may need high back corsets or braces, with abdominal support A firm mattress with fracture boards beneath it, and judiciously applied heat and massage are benefical in most instances (*See also* Chapter 34)

Hip Joint Disease.—The management of degenerative hip joint disease is not too satisfactory once well marked roentgenographic changes become manifest [illegible]

degree of success, the physician must keep certain physiological principles in mind

[illegible]

of

acc

pel

of

and other muscles develops and adduction and extensive rotation follow flexion of the hip joint. The latter result in ligamentous strain with proliferation of bone and cartilage about the hip

Treatment should be directed first at relieving ligamentous or muscular strain This is accomplished best by rest in the recumbent position. In [illegible]

As a rib sags, the costotransverse articulation is used as a fulcrum and the rib is twisted resulting in an abnormal rotation of the head. Long maintenance of faulty positions may lead to irritation and true degenerative joint involvement.

BIBLIOGRAPHY

1. Bennett, G. A., Waine, H., and Bauer, W.: Changes in the Knee Joint at Various Ages, New York, The Commonwealth Fund, 1942.
2. Breck, L. W., Hillsman, J. W., and Basom, W. C.. Ann Surg, 120, 88, 1944
3. Brailsford, J. F.: Radiology of Bones and Joints, Baltimore, William Wood & Co, 1934.
4. Burns, B. H., and Ellis, V. H.: Recent Advances in Orthopedic Surgery, Phila-
5. our Cancer, 17, 348, 1933
6.
7.
8. York, Thomas Nelson & Sons, 5, 605, 1935.
9. Fenn, G. K: Med Clin. North America, 19, 1077, 1936
10. Fischer, A. Rheumatismus und Grenzgebiete, Berlin, Julius Springer, 1933
11. Gilmour, J.: Brit. Jour. Surg, 26, 670, 1939.
12. ——— Ibid, 26, 700, 1939.
13. Goette, J. A.: Report on Chronic Arthritis, Rep. on Public Health and Med
14.
15. s of the Bones and
16. Am. Med Assn., 93, 514, 1929.
17. 948, 1942.
18. History and Cure of Diseases, 2d ed,
19. Hench, P. S. Nelson's Loose Leaf Living Surgery, New York, Thomas Nelson & Sons, 3, 101, 1935
20. ———. In Barr's Modern Medical Therapy in General Practise, Baltimore, Williams & Wilkins Company, 1940
21. Hill, Leonard: Brit Med. Jour, 2, 276, 1939
22. Keefer, C. S. Med Clin North America, 18, 947, 1935
23. Kelly, L. M. C. New York State Jour. Med, 42, 336, 1942.
24. Key, J. A. Quoted by Smith and Kountz, Ref 42.
25. Kistler, G. Arch Surg, 29, 589, 1934
26. Kleinberg, S.. Arch Surg, 39, 637, 1939.
27.
28.
29. 1941.
30.
31.
32.
33. 16, 149, 1909
34.
35. Pardee, H. E. B. Med. Clin. North America
36. Phemister, D. B. Jour. Bone and Joint Surg, 12, 769, 1930
37. ———. Surg, Gynec and Obst., 59,
38. Plewes, L. W: Brit Jour. Surg., 27,
39. Schmorl, G, and Junghanns, H: D... kranke Wirbelsaule Roentgenbild, Leipsic, Georg Thieme, 1932.
40. Seltzer, ...cine, 22, 163, 194
41. ——— , 1943.
42. Smith, J. ..., W. B.: Ann

or corset assist in decreasing the forward inclination of the pelvis and in securing weight bearing so that the head of the femur is in contact with the strong, superior portion of the acetabulum.

Conservative measures include rest from weight bearing, heat and sedative and analgesic drugs. In patients with a movable hip, extension traction applied to the lower limb with the patient recumbent on a firm mattress is the most effective measure in relieving pain and correcting the flexion and adduction deformity.[18] In ambulatory patients, crutches or a cane should be used, but several periods of recumbency each day should be insisted upon. It should be emphasized, however, that the *relief of pain usually is directly proportional to the amount of protection from weight bearing.*

Most forms of heat will aid in alleviating pain whether one uses hot fomentations, diathermy, radiant heat, or other measures. Roentgen therapy sometimes relieves pain and may produce some increase in motion It may be helpful particularly in elderly patients with severe pain. Procaine injections usually are not too beneficial, but hydrocortisone injected *intra-articularly is sometimes helpful in relieving pain.*[20]

At times, motion of the hip may be reduced to a minimum by immobilization in a plaster cast or by using a light ambulatory plaster hip spica. Some surgeons prefer a short leather and steel spica brace to reduce motion

[illegible]

and acetabulum, surgical treatment is preferred. In those individuals who are fair or good risks for major surgery, *arthroplasty and arthrodesis are the* procedures of choice. In those individuals with bilateral degenerative hip joint disease, *arthroplasty* is the only surgical procedure which can be

[illegible]

Degenerative Joint Disease of the Knees.—The general procedures outlined above usually suffice in the treatment of this condition. *Frequently, pain in the knee results from weak, painful feet so that treatment must include care of the feet.* Occasionally, free bits of cartilage or bone are present in the knee (*joint mice*). When present, they may cause considerable pain and locking and may lead to rapid and extensive degenerative changes, they should be removed surgically, preferably before degenerative changes

[illegible] changes may result from dis-

[illegible] In these cases *synovectomy* may

[illegible]

BIBLIOGRAPHY

1. Bauer, W. New England Jour Med, 221, 524, 1939
2 Boots, R. H, Yen, L C, McMorrow, K J, and Ragan, C: Jour Am. Med Assn, 147, 549, 1951
3. Brown, C Y, Jessar, R A, and Hollander, J. L · Jour. Am Med Assn, 147, 551, 1951
4 Camitz, H Quoted by Mulder, Ref 22
5 Clemmesen, S Quoted by Hench *et al*, 9th Rheumatism Review, Ann Int Med, 28, 146, 1948
Dale, M · Jour. Mich. Med. Soc, 49, 1068, 1950

43 SNOW, W B: New England Jour Med, **229**, 959, 1943
44 STECHER, R M New England Jour Med, **222**, 300, 1940
45 ———. Am Jour Med Sci, **201**, 801, 1941
46. ——— Am Jour. Roent and Rad Therapy, **59**, 326, 1948
47 SWAIM, L T.: Jour Lab and Clin Med, **15**, 1182, 1930
48 TURNER, E L, and OPPENHEIMER, A Ann. Int Med, **10**, 427, 1936.
49 Unpublished data from Ashburn General Hospital, McKinney, Texas.
50 WIBERG, G: Acta Chir Scandinav, **83**, 1, 1939
51 WILLIUS, F A: Proc Staff Meet, Mayo Clin, **13**, 11, 1938
52 ZOLLNER, F: Virchows Arch f path Anat, **277**, 817, 1930

7. DAVIS, D: Am. Heart Jour, 35, 70, 1948
7a DOWDELL, W. F, SOLOMON, W. M, STECHER, R. M, and WOLPAW, R.. Ann Rheumat Dis, 11, 168, 1952
8 EIDENOW, A · Brit Jour. Rheumat, 2, 13, 1939
9 ELLMAN, P: Post-Grad. Med Jour, 12, 211, 1936
10 FARLEY, R T Jour. Am Inst Homeop, 31, 405, 1938
11 FOX, R. F Brit Med Jour, 1, 409, 1936
12 FREEMAN, S Jour. Am Med Assn, 119, 968, 1942
13 GRAUBARD, D J., ROBERTAZZI, R W, and PETERSON, M. C.: New York State Jour. Med, 47, 2187, 1947
14 GRAUBARD, D J., KOVACS, J, and RITTER, H. H · Ann Int Med, 28, 1075, 1948
15 HALL, F C, and MUNROE, R T Jour Lab and Clin Med, 18, 439, 1933
16 HARMON, P H Penna Med. Jour., 45, 948, 1942
17 HENCH, P S: Ann Rheumat Dis, 7, 43, 1948.
18 HENCH, P S, *et al* · 9th Rheumatism Review, Ann Int. Med, 28, 128, 1948
19 HENCH, P S, SLOCUMB, C H, POLLEY, H F, and KENDALL, E C Jour Am Med Assn, 144, 1327, 1950
20 HOLLANDER, J. L., BROWN, E M, JESSAR, R A, and BROWN, C Y · Jour Am Med Assn, 147, 1629, 1951.
2[illegible]
2[illegible]
2[illegible]
2[illegible] ur
Am Med Assn, 149, 729, 1952
25 LIVINGSTON, S K · Arch Phys Ther, 17, 704, 1936
26 LUNDAR, J Am Jour Roent and Rad Therap, 66, 947, 1951
27 MAGNUSON, P. B, MCELVENNY, R. T, and LOGAN, C E Jour Mich. Med Soc, 46, 71, 1947
28 MAWSON, R. Brit Med Jour, 2, 691, 1946
29 MCCHESNEY, E W, and MESSER, F H: Am Jour Physiol, 135, 577, 1942
30 [illegible]
37 SCOTT, S G Lancet, 2, 1521, 1936
38 ——— Rheumatism, 1, 1, 1938.
[illegible] 1, 24, 1227,
44 SNYDER, R. G, and SQUIRES, W H · New York State Jour Med, 40, 708, 1940
45 STAMM, T T: Lancet, 2, 754, 1939
46 STEINBROCKER, O Arthritis in Modern Practice, Philadelphia, W B Saunders Co, 1941
47 [illegible]
48 [illegible]
49. [illegible]
50
51
52
53

CHAPTER 40

TREATMENT OF DEGENERATIVE JOINT DISEASE (OSTEO-ARTHRITIS)

By Nathan R. Abrams, M.D.

In treating an individual afflicted with degenerative joint disease, one must realize that he is dealing with a patient suffering from a *chronic* [illegible]

time required to explain it to the patient. *The treatment of each case must be individualized.* Its success depends upon the physician's understanding and the patient's cooperation.

One of the most important points in treatment is to *reassure the patient that his disease is annoying but not serious, that he does not have crippling* [illegible]

From a *pathological* standpoint it must be realized that this disease is chronic and cannot be *cured*. Since worn or damaged cartilage regenerates poorly, at best, and since osteophytes cannot be resorbed, such changes, once manifest, are *irreversible* and *permanent*. Nevertheless, much can be done to relieve symptoms and to prevent, or at least retard, progression of the pathological conditions.

In treating degenerative joint disease, the physician must outline and explain an individualized program to the patient. Without a complete program the administration of so-called "specific" drugs has little value.

GENERAL CONSIDERATIONS

Rest.—*Just as cardiac patients are advised to live within their hearts so* should patients with degenerative joint disease be advised to *live within their joints*. A joint which shows degenerative changes cannot endure much stress or strain. If it is forced to perform normal functions, *degeneration* will be accelerated. Therefore, *rest* is primarily important in the treatment of this disease.

The amount and types of rest vary with regard to the joints involved and [illegible] complete rest in bed.

ing and afternoon, in a wage-earner, a period of rest should [illegible] noon, if possible. As a rule, patients can do more, with less damage to their

joints, by dividing the day with rest-periods than by trying to work

range of motion at least several times a day

General Plan of Treatment of Degenerative Joint Disease
Adequate daily *periods of rest of the involved joints.*
Avoidance of trauma, strain or abnormal use of the affected parts
Reduction in weight if obese and especially if the lower limbs are involved, adequate vitamin intake
Physical therapy—*heat, massage* and *exercise* (properly administered)
Good Body Mechanics elimination of faulty posture, proper support and exercises for the feet.
Orthopedic appliances. support to the back, hip, knees or feet if indicated, use of corsets, adhesive or elastic bandages, crutches, canes, caliper splints, etc , as required
When necessary, relief of pain by analgesic drugs (aspirin and phenacetin).
Intra-articular hydrocortisone injection
Pr
M

Correction of Factors Causing Excessive Strain.—All factors which tend to cause excessive stress and strain on a diseased joint should be corrected if possible One of the most frequent factors in cases involving weight- e usual measures

Pronated feet, genu valgum and varum and curvatures of the spine all place extra strain upon the knees and low back. Faulty posture should be corrected and proper support prescribed for the feet, the pendulous abdomen and breasts, the lordotic lumbar spine, etc. Chronic strain may result also from one's occupation, recreations, etc.

Physical Therapy.—Most patients benefit

may be applied in many forms—baking, infra-red, hot fomentations, electric pads, short wave, micro-wave, or conventional diathermy, paraffin baths, etc. Each of these has the effect of producing an increased blood supply in the area treated. Home methods, where heat can be applied several times a day, are preferable to infrequent office treatments *Often*

PART VII

ARTHRITIS FROM DISORDERS OF METABOLISM

CHAPTER 41

GOUT

By Charley J Smyth, M.D.

Definition.—Gout is an hereditary disorder of uric acid metabolism occurring predominantly in males It is usually characterized by hyperuricemia, acute and recurring attacks of severely painful arthritis, deposits of sodium urate crystals in articular, periarticular and subcutaneous tissues, and in advanced stages by cardiovascular lesions, nephritis with urinary calculi, and frequently severe crippling. The clinical course is extremely variable and can be divided into stages based upon the frequency and

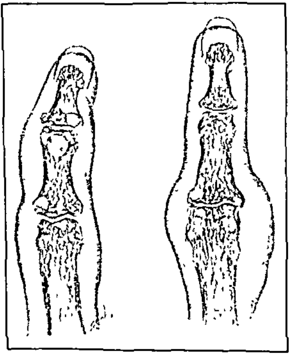


Fig. 233 —Gout Drawing shows asymmetrical swellings and punched-out areas in the phalanges

when rest is important, a patient does himself more harm by coming to an office for treatment than he gains from the treatment.

Frequently, moist heat is more beneficial than dry. In deep-seated areas (*e. g.*, disease of the spine) *diathermy* is more efficacious than most other forms. Painful Heberden's nodes usually are aided by hot soaks, (plain water or epsom salts) paraffin baths or contrast baths. Most patients benefit from warm baths, therapeutic pools and spas.

Massage can be helpful as a follow-up to heat treatments It is advisable to avoid, or at best massage very gently, the areas over joints, particularly if they are tender Irritating the tender tissues covering osteophytes may increase their size and sensitiveness. Massage of the muscles between joints may be rather vigorous to prevent muscular atrophy and weakness Moderate *exercise* of involved joints is helpful in reducing muscle spasm and muscle atrophy. Generally, exercises should be repeated three or four times daily, beginning with small amounts at each session and gradually increasing. Excessive exercise may aggravate the articular pain and stiffness. *Discomfort persisting for more than one or two hours following exercise indicates that it has been too great and therefore should be curtailed* Active exercises are preferable to passive ones as the latter may tear tissues or injure marginal proliferations

[illegible] local problem. In [illegible] pri- [illegible] been proven. The use of various drugs will be discussed below. As with other chronic diseases the patient, himself, and not just his articular symptoms, should be treated *Psychotherapy* is of great importance.

Diet.—There is no substantial evidence that diet plays any specific rôle in this disease, except in relation to obesity. As mentioned above, obesity places undue strain on weight-bearing joints. When these joints are involved in obese patients, weight reduction frequently is attended by gratifying relief of symptoms

DRUG THERAPY

Thyroid Medication.—It has been reported that many patients with degenerative joint disease show a subnormal *basal metabolic rate* (Bauer, however, noted subnormal metabolic rates no oftener than in any group of cases of similar age.[3])

Some patients are said to improve following thyroid medication.[5, 15] The dose of dessicated thyroid varies with the individual. Whether such improvement is due to the correction of the lowered metabolic status or whether it is due to weight reduction with consequent lessening of strain due to obesity is a matter of conjecture. Many individuals with lowered metabolic rates suffer from "arthralgia" (fibrositis) rather than true degenerative joint disease.

Analgesic Drugs.—These drugs are helpful in controlling pain, but must be used cautiously. Undue damage may ensure in the diseased joints as a result of masking pain. Acetylsalicylic acid and compounds of this drug with acetophenetidine are less expensive, sa[illegible] in individuals allergic to them), and frequently more efficacio[illegible] other drugs of this type. Local counte[illegible] (by methy[illegible] algesic balm,

severity of the attacks and the presence or absence of tophi. Asymptomatic hyperuricemia (larval gout) may be present for some time before acute arthritis appears This is followed by periodic episodes of a few days to several weeks of acute joint inflammation with complete remissions between bouts of acute joint swellings. In the last phase of the disease, usually ten years or longer following the initial attack, chronic gouty arthritis occurs, multiple joints are involved and tophaceous deposits are frequently found

History.—Gout is one of the oldest of known diseases. It was described as early as the fifth century B C by Hippocrates as podagra, cheiagra, or gonagra, depending on whether the big toe, wrist or knee joint was involved. Tophi were first described by Galen (131 to 200 A.D.)[90] The term gout is derived from the Latin *gutta*, a drop It was used in the thirteenth century to signify that the poison had entered the affected joint drop by drop.[62, 64]

Colchicum was known to Byzantine physicians as early as the fifth century A D under the name of hermodactyl (finger of Hermes) A drug probably identical with colchicum was described in Ebers Papyrus (1500 B.C.)[90] Baron Anton von Storch[116] introduced colchicum autumnale (or meadow saffron) to most of Europe in 1763 He was physician to Empress Maria Theresa He stressed the analgesic action of the drug for articular pains and its diuretic properties. It is reported that Benjamin Franklin introduced colchicum into this country [107]

Sydenham (1624 to 1689) was the first to differentiate gout from other types of arthritis His colorful and dynamic description of the acute gouty attacks remains unsurpassed In 1797 Wollaston discovered that the "chalk stones" in gouty joints were composed of uric acid.[139] Garrod showed in 1848 that the blood of gouty persons contains abnormal amounts of uric acid To demonstrate urate in the blood he devised a simple method known as the "thread experiment", when a thread was placed in the blood of a gouty patient, the sodium urate crystallized and adhered to the thread.

Cinchophen was synthesized in 1887 and was introduced as a drug for the treatment of gout by Weintraud in 1911.[132]

Incidence.—There is wide variation in the incidence of diagnosed gout in the different countries from which reports are available. The prevalence of gout in continental Europe[127] was less than 1 per cent; in England it was 2.7 per cent in one series[37] and 4 per cent in another;[37] in the United States of America (Mayo Clinic) 5 per cent, and in a report from India it was 7 per cent In one United States Army Arthritis Center during World War II 5200 cases of arthritis or related disorders were admitted of which 21 were

It has been found at an early age in apparently normal relatives of gouty

subjects and is a finding in some adults years prior to the onset of acute gouty attacks. It is probable that many individuals with familial hyper- ... joint symp- ... number of

Acute arthritis occurs with increasing frequency from the second to the

many ...

Sex. ...

uncom ...

male It has also been stated that men deprived of their gonads do not become gouty. Rosenberg reported a case with bisexual genitalia, male configuration of the body but with a predominantly female personality who had characteristic attacks of acute gouty arthritis [106] Claiborne reported a seventeen year old girl with pretophaceous gout of three years' duration [23] Typical tophaceous gout has been reported in women.[121,131] (Fig 234). Thomson cautioned that gout may assume atypical forms in women, especially about the menopause, and because of its infrequency in females, is thus overlooked [125] In his cases, gout occurred in a more chronic form in women, often superficially resembling rheumatoid arthritis In a recent review of 1484 cases of gout 98 (6 6 per cent) were in women.[85]

depends on differences in the excretory mechanism for urate. To explain the existence of the normal sex differential and the inherited hyperuricemia which precedes gout, this author offers the theory that in the gouty individual, whether male or female, there is an abnormal androgen of adrenal cortical origin which causes the elevated plasma urate.

Race and Climate.—Persons of all nationalities are subject to the disease Cohen has discussed gout in the negro [26] There are at present no comprehensive analyses concerning the incidence of gout among different races Gout is reported most commonly in temperate zones, it is known to occur in the tropics Gout has been reported in Chinese [149 167]

ETIOLOGY AND PATHOGENESIS

Heredity.—The familial nature of gout was recognized by the ancient Greeks and since then medical writers have stressed the importance of heredity in this disease ...

reported by Scudamore [108] In recently reported series of patients the incidence varies from 10 to 18 per cent.[64]

The exact hereditary mechanism in gout has only been established in the past few years. An analysis of data from well authenticated reports[39 57 120] by the principles of genetics and statistics leaves no reasonable doubt that gout is always an inherited disease.

pool is replaced each day, representing 700 to 850 mg. of new uric acid entering the pool each day. The rate of uric acid formation has been calculated from the rate of all of the isotope concentration and was found to exceed the rate of urinary excretion by 20 per cent or more. This evidence indicates that a portion of uric acid undergoes catabolic breakdown in man

Radioactive N^{15} uric acid has been used to study the influence of ACTH, cortisone and colchicine on the urate pool-size and turnover rate.[145] In one patient with a uric acid pool-size of 3300 mg. (three times normal), colchicine caused a 50 per cent decrease in pool-size and an increase in turnover rate of from 0.50 to 1.01 pools per day This suggests that colchicine might decrease the rate of uric acid synthesis In a similar patient, cortisone caused a decrease in pool-size of 30 per cent and increased the turnover rate from 0.59 to 1.26 pools per day. Another patient with a similar pool-size showed no response to these two drugs, he had been maintained on a small daily dose of colchicine for some time.

The effect of salicylates upon the miscible pool of uric acid in gout has also been studied using isotope technique[111] When uricosuric doses of

than in the normal man and that overproduction of uric acid is largely responsible for the pathological increase in the size of the miscible pool of

and it is not derived exclusively or even in major part from preformed purines or nucleoproteins Uric acid can also be formed from the purines of the nucleic acids It is now clear that purines and purine derivatives can be synthesized in the body from comparatively simple molecules and that

severity of the attacks and the presence or absence of tophi. Asymptomatic hyperuricemia (larval gout) may be present for some time before acute arthritis appears This is followed by periodic episodes of a few days to several weeks of acute joint inflammation with complete remissions between bouts of acute joint swellings In the last phase of the disease, usually ten years or longer following the initial attack, chronic gouty arthritis occurs, multiple joints are involved and tophaceous deposits are frequently found.

History.—Gout is one of the oldest of known diseases It was described as early as the fifth century B C by Hippocrates as podagra, cheiagra, or gonagra, depending on whether the big toe, wrist or knee joint was involved Tophi were first described by Galen (131 to 200 A D) [90] The term gout is derived from the Latin *gutta*, a drop. It was used in the thirteenth century to signify that the poison had entered the affected joint drop by drop [62,64]

Colchicum was known to Byzantine physicians as early as the fifth century A D under the name of hermodactyl (finger of Hermes). A drug probably identical with colchicum was described in Ebers Papyrus (1500 B.C) [90] Baron Anton von Storch[116] introduced colchicum autumnale (or meadow saffron) to most of Europe in 1763 He was physician to Empress Maria Theresa He stressed the analgesic action of the drug for articular pains and its diuretic properties It is reported that Benjamin Franklin introduced colchicum into this country.[107]

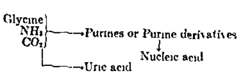
Sydenham (1624 to 1689) was the first to differentiate gout from other types of arthritis His colorful and dynamic description of the acute gouty attacks remains unsurpassed In 1797 Wollaston discovered that the "chalk stones" in gouty joints were composed of uric acid.[139] Garrod showed in 1848 that the blood of gouty persons contains abnormal amounts

Cinchophen was synthesized in 1887 and was introduced as a drug for the treatment of gout by Weintraud in 1911 [132]

America (Mayo Clinic) ... per cent, ...
cent In one United States Army Arthritis Center during World War II 5900 cases of arthritis or related disorders were admitted of which 21 were
that
s the
Until
roups

It has been found at an early age in apparently normal

they may be incorporated into nucleic acid or directly oxidized to uric acid. This over-all scheme for uric acid formation has been presented by Benedict, *et al.*[140] as follows:



Low 17-Ketosteroid.—*Urinary 17-ketosteroids are substances largely due to the metabolism of adrenocortical and testicular androgens. Decreased urinary 17-ketosteroid output has been found to be a constant finding in patients with gout*[165,177] The average is 3.2 mg. per twenty-*four hours which is about one-third of the minimal normal. This low* steroid excretion has been found to be equally low both during attacks of acute gouty arthritis and during asymptomatic interval gout. In gout patients injected testosterone was recovered as urinary 17-ketosteroids in the usual extent Since the gouty subject appears to secrete reduced amounts of normal androgens, it has been postulated by Wolfson that androgenic function is maintained by an abnormal male sex hormone from the adrenal cortex which does not make an important contribution to urinary 17-ketosteroid when it is metabolized This "gouty androgen" does not seem to be present with non-gouty hyperuricemia or in rheumatoid arthritis.

Electrolyte Changes

... water exchange associated with attacks of ... ulib- ... uric ... attacks of gout ...

in body weight on the days of increased urinary output.

PATHOLOGY

Opportunities to study the changes in bones and joints removed at operation or at necropsy from gouty subjects are few, thus our knowledge of the pathological manifestations of this disease is limited. It is generally accepted that the tissue changes appearing in gout are due to the precipitation of uric acid in the form of sodium urate in the affected part. There is no satisfactory explanation for this urate deposition.

Gross Examination.—Gross examination of the gouty joint early in the disease may reveal nothing except deposits of crystals on the surface of the articular cartilage and the central avascular portion is more frequently ... parti-

bone (Fig. 250) and in the ...

individuals from gouty families will give us the final answer

Acute arthritis occurs with increasing frequency from the second to the sixth decade, *being most common at about forty years of age.* The first evidence of the disease may not appear until late in life When juvenile gout occurs the progress is likely to be rapid with severe seizures involving many

Sex.

uncom

male.

becom

configuration of the body but with a predominantly female personality who had characteristic attacks of acute gouty arthritis [106] Claiborne reported a seventeen year old girl with pretophaceous gout of three years' duration [23] Typical tophaceous gout has been reported in women.[121,131] (Fig 234) Thomson cautioned that gout may assume atypical forms in women, especially about the menopause, and because of its infrequency in females, is

nic form in

In a recent

the existence of the normal sex differential and the inherited hyperuricemia which precedes gout, this author offers the theory that in the gouty in-

Cohen has discussed gout in the negro [26] There are at present no comprehensive analyses concerning the incidence of gout among different races Gout is reported most commonly in temperate zones, it is known to occur in the tropics Gout has been reported in Chinese [149,157]

ETIOLOGY AND PATHOGENESIS

Heredity.—The familial nature of gout was recognized by the ancient Greeks and since then medical writers have stressed the importance of heredity in this disease. A history of gout in other members of the family or antecedents can be obtained in 50 to 60 per cent of cases if the examiner persists in his questioning [126] A family incidence as high as 75 per cent was reported by Scudamore.[108] In recently reported series of patients the incidence varies from 10 to 18 per cent [84]

The exact hereditary mechanism in gout has only been established in the past few years An analysis of data from well authenticated reports[14,67,120] by the principles of genetics and statistics leaves no reasonable doubt that gout is always an inherited disease

be so s as in 1 such instances Bony trabeculae may be destroyed following invasions of the subchondral bone space by the pannus This leads to the punched-out areas noted on the roentgenograms (Fig. 237) Owing to the joint destruction, osteophytes may be produced along the margins of the involved joint, the subchondral bone becomes sclerotic and changes of secondary degenerative joint disease are added to the changes caused by the chemical irritation.



Microscopic Examination.—For specific demonstration of the characteristic urate crystals gouty tissues require special handling. If fixed with formalin and sectioned no crystals will be seen but merely the clefts from which the urates have been dissolved. Techniques have been described for the preservation and microscopic demonstration of urates in gouty tissues [32, 34] With the de Galantha method absolute alcohol is used as the fixative.

The urate deposits in soft tissue are surrounded by an inflammatory reaction with a connective tissue capsule and many large multinucleated foreign body giant cells [110] The deposits are seen embedded in the articular cartilage, bone marrow, synovial membranes, joint capsules, ligaments, periosteum, tendons, bursae, and in the subcutaneous and intramuscular tissues. No joint is exempt from urate deposition, however the metatarso-

Shortly after the development of quantitative methods for determining the uric acid content of the blood, Folin and Lyman[40] reported hyperuricemia in a normal man, many members of whose family had been gouty. Jacobson[67] studied a son of each of two men with clinical gout and a brother of a third patient; all of these persons had elevated blood urates. [illegible] of 136 non-gouty relatives of 27 [illegible] relatives (25 per cent) had hy[illegible] [illegible]e persons with the elevated levels to the patients is unfortunately not given in this paper, but in the two pedigrees which were published, all of the individuals with hyperuricemia had a parent with similar findings, whenever the parents were studied. Smyth and Freyberg[111] have reported similar findings in a study

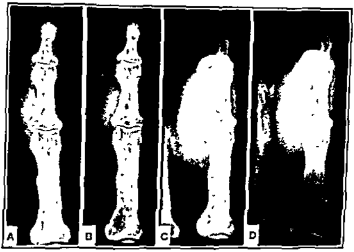


Fig. 233 —Middle finger of a woman eighty-two years old with gout showing progressive erosion of phalanx and overlying tophus Earliest changes on left (*A*), one year later (*B*), four years later (*C*), after five years (*D*)

of all of the immediate relatives of two gouty kindred Data from a more extensive study by these authors of 87 relatives of 19 gouty families have been analyzed from the genetic standpoint by Dr. C. W. Cotterman The pedigree of one of the families in this study is shown in figure 234 The results in this study show that hyperuricemia in gouty families is apparently due to a single dominant autosomal (that is, not sex-linked) gene Only a portion of heterozygotes for this gene develop recognized gouty arthritis Stecher[115] investigated 203 members of 40 gouty families based on 248 serum uric acid determinations The genetic mechanism involved in the inheritance of gout suggested by these workers is that the gene involved is an autosomal dominant with a much lower penetrance in the female than in the male [illegible]

sexes, with heterozygous males having on the average higher [illegible] levels than heterozygous females A possible explanation of the greater

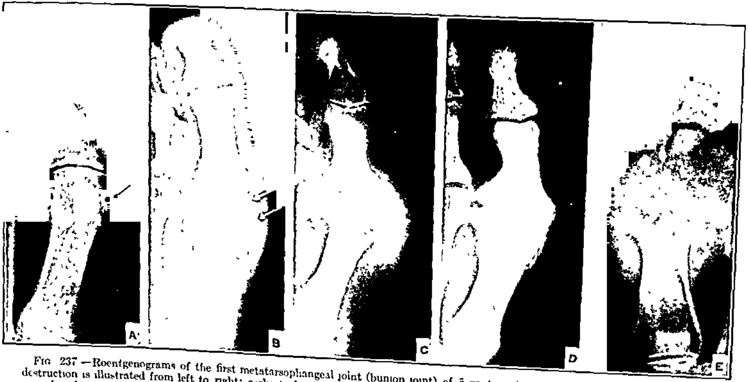


FIG 237 —Roentgenograms of the first metatarsophangeal joint (bunion joint) of 5 gouty patients Progressive erosion and joint destruction is illustrated from left to right; earliest change is a punched-out area (A), more marked destructive changes (B and C), secondary hypertrophic changes (D), far advanced destruction (E).

incidence of clinical gout in males than in females is that this disease tends to develop in those persons of either sex whose mean uric acid levels exceed a certain value for a certain period, and since hyperuricemic males tend to have higher values than hyperuricemic females, more males than females have gout The carriers are those individuals with the postulate dominant, and hence hyperuricemia, who do not develop, or have not yet developed, gout.

All of [illegible]
individua[illegible]
attacks c[illegible]
[illegible]

menopause.

The Rôle of Hormones in Pathogenesis.—In suitable dosage ACTH will terminate a spontaneous gout attack within 24 hours [52,152,161,164,166,171] However, when the hormone is subsequently discontinued most patients will experience a recurrent attack within the following few days ACTH given to a patient in the interval stage of gout may precipitate acute gouty arthritis [52,152,164] The onset of joint symptoms usually begins within 96 hours after ACTH withdrawal. In other words, ACTH tends to stop an attack which is in progress and to produce an acute attack in interval gout by ACTH withdrawal.

[illegible]

ACTH which stimulates the adrenal cortex to increase its output of steroid hormones and thus to correct the corticoid-lack. The gouty individual fails to restore a normal adrenal cortical status when a state of lack of adrenal glycocoids is induced by the withdrawal of exogenous ACTH.

The known incidents of acute gouty arthritis are situations which increase the requirement of 11-oxysteroids (*e g.* surgical operation, myocardial [illegible]

[illegible]

ministered orally, intravenously or intra-articularly will not cause gouty symptoms in the normal or gouty subject. Acute gouty attacks commonly occur in joints that do not have roentgen evidence of urate deposits. There are no significant changes in the serum urate level [illegible]
of [illegible]
[illegible]
gr[illegible]
sh[illegible]
excruciating pain [175] Striking amplitude increases with blood flow and oscillometer indices up to 600 per cent of normal have been recorded. After ACTH the vascular abnormalities rapidly return toward normal, within a

phalangeal joints of the great toes (bunion joints) are most frequently involved Next in order of frequency are the intertarsal joints of the feet, ankles, fingers and wrists Urate deposits are rarely found in the sacro-iliac, temperomandibular, sternoclavicular articulations, spine, shoulders or hips.[90] In rare instances tophi have been found in heart muscle,[11] tongue, bronchi, pericardium, pleura, meninges, and cornea A case of a painful stump has been reported in which the discomfort was attributed to the pressure of minute uric acid crystals against nerve endings[59] Treatment of the gout resulted in relief of symptoms.

The kidneys frequently show punctate dots of urate crystals in the cortex and as linear streaks in the medulla In addition there is varying degrees of glomerular fibrosis Those who have studied the renal lesions of this disease consider that it is primarily a vascular sclerosis.[8,107] The incidence of vascular sclerosis is higher in patients with gout than in people of the same age without gout.[8] The immediate cause of death is usually coronary or cerebral vascular disease or nephrosclerosis with uremia

CLINICAL COURSE

Initial Gouty Attacks.—Acute gouty arthritis usually begins in men over thirty years of age. The attack is usually of sudden onset, frequently at

awakening.

The dramatic suddenness of the onset of an attack of gout is almost diagnostic The affected joint, often in a metatarsophalangeal, may swell markedly within a few hours and become hot, dusky red and extremely tender This swelling usually extends for some distance beyond the margin of the joint and is more marked than in other forms of acute arthritis Not uncommonly, there may be effusions into the large joints During the acute bout of pain the involved joint, usually the foot, resembles an extensive cellulitis with its tense, red or cyanotic shiny skin and distended superficial veins with the inflammatory edema extending far beyond the affected joint With subsidence of the redness and tenderness, pitting edema can be observed The skin overlying the lesion becomes thin and loose and may itch and desquamate

The sudden onset of excruciating bouts of pain in an apparently healthy middle aged man frequently involving the basal joint of the great toe with complete freedom of joint symptoms between attacks is almost pathognomonic of gout

Joints Involved—The first attack of gout is usually monarticular involving most commonly the big toe, instep, heel, ankle or knee.[55] The smaller peripheral joints appear to be more vulnerable to attack than the knees, elbows, hips and shoulders. The big toe is affected in from 53 to 70 per cent of gouty individuals in the initial attack of the disease and is involved sooner or later in 90 to 95 per cent of these patients. The ankle was first affected in 13 and the foot in 12 of the 100 cases reported by Haden.[89] Although no joint is exempt from gout, the joints of the lower extremities

few hours, blood flow and pulse amplitude may decrease 50 per cent. These observations suggest that the acute attack of gout is characterized by vasodilitation in the area with clinical manifestations, and that acute arthritic symptoms are principally of vascular origin.

Mechanism of Hyperuricemia.—The exact mechanism for the increased uric acid content of the blood or the cause for the precipitation of sodium urate in the tissues of patients with this disease is unknown. Three possible mechanisms have been investigated to explain the hyperuricemia which occurs in the gouty individual. The increased concentration of uric acid is considered by some to be a result of increased formation; others consider the increased concentration of uric acid to be the result of some abnormality in its renal excretion; others attribute the hyperuricemia to a decreased destruction of uric acid.

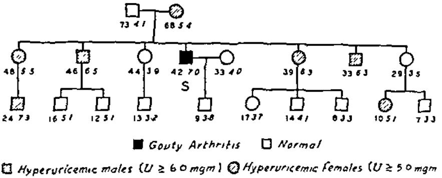


FIG. 235.—Pedigree of a gouty male patient (black square). The squares represent male relatives and the circles females. Figures below the pedigree symbols represent age in years, followed by serum urate concentration (U) in Mg. per cent.

Other Theories.—The cause of episodes of acute gouty arthritis remains unknown. It has not been proven that they result from, or are associated with, urate deposition in joints, nor that variations in the degree of hyperuricemia bear any consistent or causal relation to acute attacks. Some workers feel that the disease may be produced by any of the changes causing an alteration in the chemistry of the tissues favoring precipitation and fixation of uric acid salts.[91]

One of the theories concerning gout is that the onset is marked by an increase of the joint fluid with a deposition of sodium urate crystals in the fluid. These crystals adhere to and erode cartilage and replace the destroyed portion. The capsule and periarticular tissues also become involved and in the later stages, the urate crystals may replace portions of the ends of the bones and of the marrow. A similar pathological picture may occur in the subcutaneous tissues, muscles, bursae, cartilage, or in the helices of the ear. Deposits of uric acid crystals may also occur in the kidneys and lead to uric acid stones.

... have emphasi... e of allergy in gout.[24,48,92]
n allergen in food, bacteria
or operation).

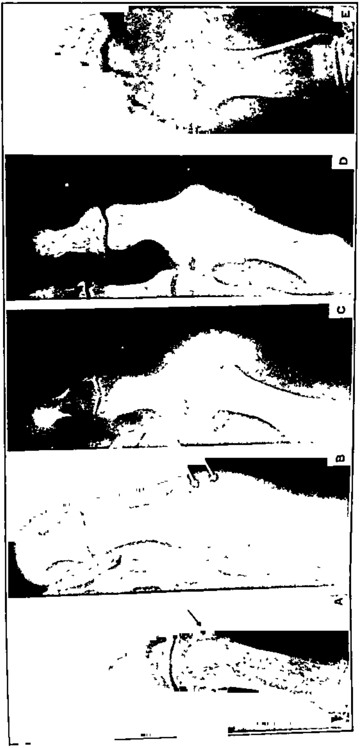


Fig 237 —Roentgenograms of the first metatarsophalangeal joint (bunion joint) of 5 gouty patients. Progressive erosion and joint destruction is illustrated from left to right; earliest change is a punched-out area (A), more marked destructive changes (B and C), secondary hypertrophic changes (D), far advanced destruction (E)

URIC ACID METABOLISM

Emil Fisher in 1907 established the fact that, in the human, uric acid is the end product of purine metabolism, and since that time the rôle of purine

to the uric acid problems in normal man or the gouty individual.

Cell nuclei of all living tissues contain a high concentration of nucleoproteins which, on hydrolysis, are split into a protein and nucleic acid. Nucleic acid on further hydrolysis yields purines and pyrimidines, a sugar (d-ribose) and phosphoric acid. In birds and reptiles, uric acid represents the chief product of protein catabolism. In man, uric acid is the major end product of the metabolism of purines. The final product of protein catabolism is urea Recent application of isotope technics to the basic metabolism of gout has added much new knowledge to this aspect of the gout problem

Uric Acid.—In the blood, uric acid is present largely in the form of sodium urate; a part of the uric acid is free and part in organic combination[10 21] Wolfson[134] suggests that a large part of the uric acid in the blood exists as polymeric urate complexes Aldersberg *et al.*[2] using ultrafiltration methods, reported the existence of a uric acid protein complex in the blood of normal subjects and of patients with gout These observations were

perer[8] Further investigations will be required to establish the place of uric acid partition in gout

Normal Values

met

ave

complex tungstate reagents to a colored compound and, to assure accurate

Benedict procedures and reported a difference between the two sexes. Bulger and Johns,[21] using a procedure with *uricase and ferricyanide*, reported values for females which averaged 1 mg per cent lower than the male. Two recent additional studies[112 115] furnish evidence which indicates a significantly lower mean uric acid level in female subjects

Jacobson[47] found the uric acid content of the *serum*, collected anaero-

of uric acid

phalangeal joints of the great toes (bunion joints) are most frequently involved. Next in order of frequency are the intertarsal joints of the feet, ankles, fingers and wrists Urate deposits are rarely found in the sacroiliac, temperomandibular, sternoclavicular articulations, spine, shoulders or hips[90] In rare instances tophi have been found in heart muscle,[13] tongue, bronchi, pericardium, pleura, meninges, and cornea A case of a painful stump has been reported in which the discomfort was attributed to [illegible][85] Treat-

[illegible] als in the cortex and as linear streaks in the medulla. In addition there is varying degrees of glomerular fibrosis. Those who have studied the renal lesions of this disease consider that it is primarily a vascular sclerosis[8,107] The incidence of vascular sclerosis is higher in patients with gout than in people of the same age without gout[8] The immediate cause of death is usually coronary or cerebral vascular disease or nephrosclerosis with uremia

CLINICAL COURSE

Initial Gouty Attacks.—Acute gouty arthritis usually begins in men over thirty years of age The attack is usually of sudden onset, frequently at night, lasts from three to eleven or more days and the symptoms then disappear completely with restoration of normal joint function. Although the attacks may begin in the early hours of the morning, the symptoms are usually first detected when the individual puts his foot on the floor after awakening

The dramatic suddenness of the onset of an attack of gout is almost diagnostic The affected joint, often in a metatarsophalangeal, may swell markedly within a few hours and become hot, dusky red and extremely tender This swelling usually extends for some distance beyond the margin of the joint and is more marked than in other forms of acute arthritis Not uncommonly, there may be effusions into the large joints. During the [illegible]

joint With subsidence of the redness and tenderness, pitting edema can be observed The skin overlying the lesion becomes thin and loose and may itch and desquamate

The sudden onset of excruciating bouts of pain in an apparently healthy middle aged man frequently involving the basal joint of the great toe with complete freedom of joint symptoms between attacks is almost pathognomonic of gout

Joints Involved.—[illegible]

[illegible] elbows, hips and shoulders [illegible] cent of gouty individuals in [illegible] sooner or later in 90 to 95 p[illegible] affected in 13 and the foot in 12 of the 100 cases reported by Haden.[49] Although no joint is exempt from gout, the joints of the lower extremities

Occurrence of Hyperuricemia

Hyperuricemia is almost invariably present in patients with gout.[8] It is

not made

determina-

in 98 per

cent the value exceeds 6 mg. per cent. A few workers[49,50,85] report that the blood uric acid is not always elevated during the first attack. According to Hench,[69] the level is elevated at some stage of gout in about 75 per cent of patients. Since patients with acute gout frequently take salicylates or cinchophen for pain and because these drugs are known to reduce the blood uric acid content 50 per cent in twenty-four hours, it appears likely that in some instances where low values for uric acid have been reported the patients may have been taking one of these uric acid diuretic drugs

Elevation of blood uric acid may occur in a variety of pathological states including gout, acute and chronic glomerulonephritis, following liver therapy in pernicious anemia, cardiac decompensation, leukemia, polycythemia, lead poisoning, acute febrile diseases (including pneumonia, typhoid fever, erysipelas, scarlet fever and tuberculosis); after poisoning by ammonia, barbital, carbon monoxide, and methyl alcohol; during periods of regeneration of blood, after hemorrhage or hemolytic icterus and occasionally in patients with diseases of the liver and biliary system; severe diabetes mellitus (especially with acidosis) and sometimes in chronic eczema, psoriasis and urticaria[17] These diseases present no differential diagnostic problem in relation to gout.

Mechanism of Hyperuricemia.—The exact mechanism for the increased uric acid content of the blood or the cause for the precipitation of sodium urate in the tissues of patients with this disease is unknown. Three possible mechanisms have been investigated to explain the hyperuricemia which occurs in the gouty individual. The increased concentration of uric acid is considered by some to be a result of increased formation; others consider the increased concentration of uric acid to be the result of some abnormality in its renal excretion, others attribute the hyperuricemia to a decreased destruction of uric acid. From a study based upon isotopic (N^{15}) glycine, Benedict[140] believes that an "overproduction" of uric acid is largely responsible for the pathological increase in gout This view also derives support from the absence of demonstrable impairment of uric acid excretion in gouty subjects.

are involved more frequently than the upper.[18,88,108] The hip joint becomes involved at some time in about 2 per cent of cases of gout.

Tophi
Occur in from 21 to 48 per cent of all cases *of gout*
Are the only pathognomonic sign of gout.
Are found in the helices or antehelices of the ear, knuckles, hands, toes, feet, other joints, cartilages of the eyelids and nose, *heels,*[23] *subcutaneous* tissues or muscles, tendons of the fingers, Achilles tendon, patella,
the prepuce of the penis and the corpus cavernosum[8]
Are painless (except when they are the site of an acute attack, or interfere with motion)
Reveal, when opened, crystals of *sodium monourate, giving a positive murexide* test
Are easily overlooked unless gout is suspected
May appear white, cream colored or yellow.
Rarely appear before the tenth year of the disease except in very severe cases; do not wait until tophi are present to diagnose gout as the disease will usually have existed five or ten years before they are seen
May discharge a chalky mixture if the overlying skin breaks down.

Differential Diagnosis.—The clinical manifestations of the first attack of acute gouty arthritis may occasionally be seen in other diseases, as acute ... rheumatoid arthritis, acute ... s, intermittent hydra- ... rthritis with ulcerative

The most commonly mistaken conditions are. acute rheumatic fever, gonorrheal arthritis, and atypical rheumatoid arthritis. However, the patient with acute rheumatic fever is usually young, whereas first attacks *of gout occur more often after the age of forty.* Migratory polyarticular involvement is much more common in rheumatic fever than in gout. In younger persons, if multiple joints are involved, the fever, leukocytosis, and increased sedimentation rate may cause confusion with *rheumatic* fever; however, the absence of *cardiac involvement, epistaxis and tachycardia* favor the diagnosis of juvenile gout. Following the subsidence of the gouty inflammation, the skin often peels over the affected joints; this does ... to rheumatic fever.

... its recurrent attacks. However, ... from those with gout. In gonorrheal arthritis a genito-urinary involvement (primary or recurrent) can often be demonstrated *or obtained* by history. Gonorrheal arthritis is usually polyarticular at the onset involving several large joints but usually ... in only one or two of those affected; gout in the early stages attacks

Carriers of genetic hyperuricemia have normal values until puberty, if male, and until the menopause, if female. Wolfson[174] suggested that a separate androgen mechanism regulates the transformation of latent in-

Methods for Uric Acid.—The method of Folin[33] and that of Benedict[11] are the most commonly employed for the determination of uric acid and with care in the preparation of the reagents and frequent control determinations reliable results are obtained. Neither are specific for uric acid and unrelated non-uric acid reducing substances have been shown[15, 20, 21] to be a source of considerable error in the determination of uric acid. New methods using the specific enzyme, *uricase*, corrects for the error due to non-uric acid chromogen. Normal blood contains approximately 2 mg. per cent of uric acid by the uricase method. Klemperer[71] in 1945 described the mechanism for the destruction of uric acid by uricase. In studying the residual chromogenic material in the blood which was not destroyed by uricase, Wolfson[135] was of the opinion that the material consists of unknown purines of endogenous origin. He also analysed the contents of a gouty tophus and found it contained no non-uric acid chromogen. Using the uricase method it has been shown[104, 135] that urate diuretic drugs (cinchophen and salicylates) increase the output of chromogen as well as that of true urate. There is no reason at present to use this method as a routine diagnostic procedure. It is apparent, however, that by this enzymatic method additional knowledge of the pathogenesis of gout can be expected.

Factors Influencing Uric Acid Excretion

Renal Excretion of Urates.—Opinion on the problem of how urate is excreted is divided, but the available evidence would appear to favor the view that practically all the urate in plasma is in filterable form and that the amount of urate filtered can be considered to be the product of filtration rate and plasma urate concentration.[143] In the normal subject, this would mean 5 or 6 mg. of urate are filtered each minute. Something under 10 per cent of this amount normally is excreted in the urine. Obviously, there must be some mechanism for reabsorbing urate from the tubular urine. Talbott implies that there is an active mechanism for re-absorbing urate, but does not comment on its capacity nor on the possible rôle of such tubular re-absorptive capacity in regulating either urate excretion or the plasma urate concentration. Wolfson has presented data to indicate that there is normally little or no tubular re-absorption of urate. In his studies all of the urate passing the glomerulus appeared to be excreted in the urine. This appeared to depend upon the fact that only a small portion of the plasma urate is freely diffusible through the human glomerulus. The theory that the uric acid filtered at the glomerulus in man is close to that actually

that the gouty person excretes
Brøchner-Mortensen[147] found the
day in 11 patients with gout as

Recovery from attacks of atypical rheumatoid arthritis may be so complete and so rapid as to make one consider the possibility of gout. However, the typical patient with rheumatoid arthritis exhibits a slender build, undernourishment, symmetrical involvement of joints, fusiform joint

therapy, a family history of gout, and the later course of the disease. Effusions into joints develop in both diseases and are apt to make the differentiation of the two diseases difficult.

In gout, any portion of the foot, especially the dorsum, may be involved, resulting in swelling of the foot with redness increased, heat and tenderness. The foot resembles a cellulitis and by mistake may be incised with the intent to release pus.

Subsequent Gouty Attacks.—Subsequent to most of the initial attacks of acute gouty arthritis there is complete restoration of function in the affected part and practically all attacks follow the classical type. The intervals between attacks last for a variable time but usually within a year or two similar attacks occur which again clear entirely, leaving no residual joint abnormality. In exceptional instances, attacks may be more than thirty years apart, it is not uncommon to observe attacks eight to ten years apart with absolute disappearance of all joint phenomena between attacks. These periods become progressively shorter until acute bouts of arthritis occur several times each year, often with a certain regularity. In some patients, for unexplained reasons, the joint symptoms occur each spring and fall.

The recurrent attacks may or may not affect the joint initially involved or joints previously involved. Usually in subsequent attacks the intensity of the joint symptoms becomes more severe. Although the first few attacks are usually afebrile and most often involve only one joint, later attacks may be accompanied by a temperature of 101° to 102° F. or more and usually affect several joints.[56] Some individuals may experience indigestion, nausea, anorexia, aching, stiffness, polyuria and nocturia. Premonitory symptoms may be lacking prior to acute attacks of gout.

The disease may be polyarticular (about 5 per cent of cases) with a fulminating course from the onset; this is more likely to occur in young

at extremely frequent intervals the joints are no longer free from pain between attacks and are stiff, enlarged, and often deformed. This is the chronic arthritic phase of the disease. There is usually polyarticular involvement of the joints of the hands, feet, ankles, wrists, knees and elbows. (Fig. 238)

Tophi.—Deposits of urates (tophi) in articular, periarticular, bursae, and subcutaneous tissues are found in from 21 to 48 per cent of cases.[58] They vary in size from 1 mm. to several cm. and tophi the size of a baseball (7.3 cm.) have been observed.[25] They are seen most frequently upon the helix or antehelix of the ear (Fig. 239); they occur less often in the olecranon (Fig. 238) and prepatellar bursae, and tendons of the fingers and wrists,

compared with a mean of 374 mg. per day in 24 normal persons. In another study[151] the mean daily excretion in five gouty subjects was 561 mg. as compared with a mean of 390 mg. in 6 normal persons. Similar slightly higher values were reported by another group,[180] 523 mg. for 31 gouty subjects as compared with 423 mg. in 10 non-gouty.

It has been shown that certain drugs increase the urate output and decrease its plasma level, these substances have been called uricosuric drugs. Cinchophen;[14,28,45,136] large doses of salicylates,[43,72,136] salyrgan;[14,28] diadrast;[136] caronamide;[137] Benemid, and the hormones cortisone, ACTH, and Compound F, all effectively increase uric acid excretion. It has been *demonstrated that salicylates, cinchophen*[104,138] and caronamide[137] increase chromogen excretion as well as the true urate excretion. Quick[102] showed

requires an adequate supply of antiketogenic material

Benemid.—A new compound, "Benemid," (p-[di-n-propylsulfanyl] benzoic acid) administered orally, has been demonstrated to increase the urinary uric acid excretion in patients with gout. It was first introduced into clinical medicine by Boger[146] to produce enhancement of plasma concentration of both penicillin and para-aminosalicylic acid (PAS). In divided doses from 1 to 2 gms. per day in patients with gout, an increase in urinary urates varying from 30 per cent[163] to 58 per cent[180] has been reported. Associated with this increased urinary excretion, there is a decrease in the

the mid

appears to be a relatively safe drug, however gastro-intestinal upsets and skin rashes have been reported When there is renal impairment or a history of uric acid calculi, alkalinization of the urine is advised if this uricosuric agent is used. When aspirin is given simultaneously with Benemid, the action of the latter in lowering the serum uric acid level is completely annulled, but is restored when aspirin is discontinued[183]

Effect of ACTH and Cortisone.—One of the consistent biochemical changes produced in man by adrenocorticotropic hormone is a decreased serum urate level and a corresponding increase in urinary excretion of uric acid.[52,164,171] The excretion of uric acid may be increased as much as 80 per cent and the blood urate concentration decreased as much as 30 per cent by ACTH administration In the normal man the decreased uric acid blood level produced by ACTH suddenly rises several days after it is stopped; this rebound of urate blood level does not occur in the gouty individual. Compound E (cortisone) and Compound F also increase the urinary excretion of uric acid.

Isotope Studies and Urate Metabolism.—New knowledge resulting from the application of isotopic techniques has clarified some basic aspects of uric acid metabolism. Isotopically labeled uric acid using N^{15} has been injected intravenously in normal and gouty subjects.[142,144] Following the injection, the concentration of the isotope in the uric acid isolated from the urine can be determined and in this way the magnitude of the pool of miscible uric acid in the body as well as the turnover rate of this pool

toes, ankles, heels and in bone. Tophi have been found in the mitral valve,[22] cardiac muscle,[67] sacro-iliac joints and vertebrae.[7]

In the late stages of the disease, tophi are responsible for the knobby deformities over joints. In bursae and in subcutaneous deposits, acute inflammatory reactions occasionally result. The skin overlying large subcutaneous tophi is frequently highly vascular, thin and orange colored. It may ulcerate and discharge a white, chalky material having the consistency of toothpaste, which
needle-like crystals o
come infected. Any
and its contents examined, both microscopically and by the murexide chemical test.

Wolfson has summarized the clinical findings in 18 women with gout[173] and concludes that there is a marked difference between tophaceous and pretophaceous gout in the female. In the first place, tophaceous gout in

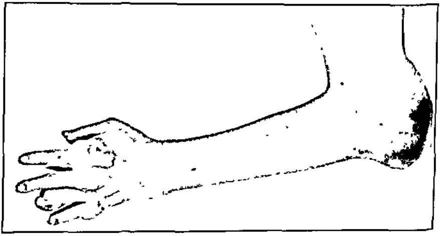


FIG 238.—Chronic gouty arthritis with shortening of the fourth finger, subcutaneous urate deposits over the knuckles and extensive olecranon bursitis.

women begins before the menopause and is associated with more impairment of renal function and higher levels of uric acid. In his study a family history of gout was obtained in half of those with tophaceous gout, which suggested to this observer that the earlier onset and greater severity of the tophaceous type was due to genetic factors.

Tophi are sometimes mistaken for Heberden's nodes, subcutaneous nodules of rheumatic fever or rheumatoid arthritis (Figs. 238 and 239), deposits of calcium, sebaceous cysts and ganglions. Periarticular calcium ... in the neighborhood of joints.[27]

Uric acid stones or gravel may be present in 10 per cent or more of patients with gout. Nephrosclerosis with deposits of urates in the parenchyma of the kidneys occurs with about the same frequency. The association of renal impairment or renal stones, with joint symptoms, should arouse a

nephritis. In the 38 gouty subjects without nephritis the kidney function

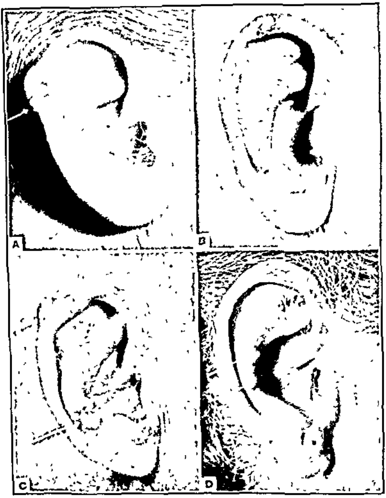


FIG. 239.—Tophi in ears. Large cystic tophus on the helix (A), ulcerated tophus (B), two tophi in ear, upon the helix and antehelix (C), subcutaneous nodules of rheumatoid arthritis, *not* gouty tophi, in a female patient (D)

was essentially normal. Brown and Mallory[111] studied five proven autopsied cases and reported that every kidney had urate deposits in the medulla

striking benign nephrosclerosis.

named Colchis.

Colchicine tablets, each 1/120 or 1/100 grain (0 53 or 0 65 mg.) should be given 1 tablet every hour or two hours until pain is relieved or until diarrhea, nausea or vomiting occurs [7,14,31] The average attack of gout requires approximately 10 tablets of colchicine (1/120 gr. or 0 5 mg.) and varies from 8 to 16 tablets Many patients learn by experience the optimum number of tablets to take with each attack, so that alleviation of joint pain may be attained without undesirable gastrointestinal symptoms. Paregoric may be necessary to control the toxic symptoms. Once the amount of colchicine necessary to produce toxic symptoms has been established, a patient may be able in subsequent attacks to obtain a satisfactory effect by taking 1 or 2 tablets less in a course. The patient should always carry colchicine and start taking the pills at the first appearance of symptoms of a gouty attack. Delay of a few hours in the treatment after the onset of acute gouty pains may increase the severity and prolong the duration of the acute episode

colchicine was reported.[32]

The response to this drug is often dramatic. Joint pains and swelling begin to subside approximately twelve hours after the institution of therapy

Treatment of the Acute Attack of Gout

Rest in bed with elevation of the affected part, if possible, no weight bearing until acute pain and tenderness have disappeared if the lower extremities are affected

Temporary immobilization of the involved part by light *splinting* and protection from bedclothes by the use of a cradle.

Colchicine, 0 5 mg ($\frac{1}{120}$ gr) by mouth every hour until pain is relieved unless toxic symptoms appear (nausea, vomiting and diarrhea); it is usually not necessary to exceed 8 to 10 doses of this drug during the first twenty-four hours

Acute Episcleritis.—Ocular inflammations associated with gout are not numerous but such cases often present a perplexing problem. They are reported to improve following the reduction of blood uric acid.[73]

Chalk Gout.—This condition is more commonly called calcinosis circumscripta and may present swellings over joints which *simulate* gouty tophi In circumscribed calcinosis, these *concretions of calcium phosphate and* calcium carbonate are confined almost exclusively to the toes, fingers, elbows and knees.[27,105] The subcutaneous calcium deposits may or may not [illegible] nearby joints. Calcium [illegible] neralization of bone or [illegible] sidiously and is of un- [illegible] Calcinosis in chapter on [illegible]

DIAGNOSIS

The diagnosis of gouty arthritis in the initial or second attack is easy in the majority of cases. Usually it involves one joint in a male patient. It is the only arthritis which comes on with such unusual rapidity and commonly after some characteristic provocative, such as minor trauma, dietary indiscretion, surgical operations or strenuous exercise. The excruciating pain is followed quickly by redness and swelling of the joint and surround- [illegible] a few days with [illegible] is an increased [illegible] s with complete [illegible]

[illegible] Olecranon bursitis is frequent with gout and should [illegible] osteoarthritis, post- [illegible] arthritis. Tophi are [illegible] ous nodules of rheu- [illegible] ium, sebaceous cysts [illegible]

Laboratory [illegible] tests are useful in the diagnosis of gout. [illegible] serum urate is of primary importance. [illegible] Any lesion suspected as a tophus should be needled or [illegible] amined for the presence of urates by microscope and the murexide test. X-ray examination is of value in the diagnosis of the late stages of gout. The changes most suggestive of gout are well-defined, punched-out areas located in the subchondral bone of the base or head of the phalanges. [illegible]

[illegible] returns to normal [illegible] other evidence of decreased renal function may occur.[97,107] Anemia is seldom present in patients with gout except at times in the late stages of [illegible][42]

and in most instances pain is completely relieved in from one to two days Over 95 per cent of acute attacks are materially benefited by a single course of colchicine Should no benefit occur a second course of colchicine should be given after an interval of three days, repetition of a course sooner might [illegible]

because it offers more exact dosage, gives quicker relief and does not cause *nausea, vomiting or diarrhea. These observations have not yet been* confirmed

The mechanism of action of colchicine is unknown. Colchicine does not stimulate the adrenal cortex, studies to establish this point have included the 17-ketosteroid output, 11-oxycorticoids and the blood eosinophile counts [165,166] It is the active ingredient in the wine and tincture of colchicum which may be of varying potency and less consistently effective than the alkaloid colchicine

ACTH and Cortisone.—Hellman in 1949 was the first to show that [illegible] ACTH [52] Rapid [illegible]

treatment of an acute gouty attack consists of the administration of 50 mg. ACTH intra-muscularly every six hours; usually no more than 3 doses [illegible]

using 1.5 mg. per day. On this combined hormone colchicine [illegible] attacks are usually ended within 24 hours with an average dose of 100 mg of ACTH In chronically colchicinized patients or those previously treated with colchicine without success, amounts of ACTH up to 120 mg. may be required.

Cortisone has been used in the treatment of acute gouty arthritis, but it has not been as successful as *ACTH* [156,158] *Doses up to 300 mg. per day are* required for the control of symptoms, failures are frequent and some patients suffer intensification of their signs and symptoms during cortisone medication.

Most observers who have used ACTH and cortisone in the treatment of acute gouty arthritis, agree that colchicine as it is customarily used will control the great majority of [illegible] ks and that for those patients who fail to respond to colchicine, [illegible] good adjunctive measure.

Laboratory Findings

Increased blood *uric acid* (*almost invariably present*)
Sedimentation rate usually normal between attacks, frequently elevated during attacks[83]
Roentgenograms generally negative early, of little use in diagnosis until late in the disease, roentgen-rays of the feet usually show signs of gout earlier than the hands. *Roentgen-rays show areas of erosion in only 32 per cent of cases*
Mild or moderate *leukocytosis* is the rule during attacks
Anemia is seldom present in patients with gout except perhaps in the terminal stages[83]
Decrease in *renal function* may occur[37,107]

Main Symptoms and Signs of Gout

Tophi present in from 21 to 48 per cent of cases at any given time[83]
Elevation of *blood uric acid* almost invariably present
Acute *recurrent attacks with complete remissions* (before the development of secondary changes in joints).
Predominance in males (95 per cent of cases)
Involvement of big toe in initial attack in 70 per cent of cases, and sooner or later in 90 per cent
Roentgen-rays of *little diagnostic value early* in the disease, bony erosions seen in only about one-third of cases even in the later stages

Axioms of Hench, Suspect Gout If

Acute joint symptoms follow an *operative procedure*
There is a *family history* of gout
There is a history of *olecranon bursitis* or Achilles tendinitis
Acute joint symptoms follow slight trauma, or excessive holiday food or drink
Sudden joint symptoms appear during early morning hours
Acute arthritis occurs in men over the age of forty, in the absence of acute trauma or a history of gonorrhea
Acute joint symptoms develop within minutes or a few hours
Joint symptoms are *associated with a chronic nephritis or renal colic*
Joint symptoms occur in patients with *polycythemia, leukemia* or after certain therapeutic procedures such as injection of liver extract, salyrgan, insulin, ergotamine tartrate (gynergen), thiamin chloride, decholin (dehydrocholic acid) or the use of a ketogenic diet
A long automobile ride or strenuous exercise precedes the first attack
Joint symptoms occur after hunting and fishing expeditions
Joint symptoms recur in the spring or fall.
Joint pain is exceptionally severe
Acute *involvement of the great toe occurs*
The patient appears wearing a shoe cut out over the metatarsophalangeal joint of the great toe (sign of the split shoe).
Complete recovery of an acute arthritis occurs within several weeks
Symptoms are *relieved* within twenty-four to seventy-two hours by adequate doses of *colchicine*.
The patient is absolutely *free from joint symptoms between attacks*
Periarticular fistulas or ulcers are present.
The symptoms in a monarticular arthritis are entirely disproportionate to the findings.
The general reaction, especially the fever in any polyarthritis, is less than the findings might indicate[124,134]

begin within a few hours and the majority of patients experience a complete remission in 48 hours or less. Marked analgesia may become apparent within a few minutes following intramuscular administration of phenylbutazone. It has been shown to be effective in patients resistant to colchicine and sometimes in patients previously treated with colchicine the response was better to phenylbutazone. Where serial determinations of plasma uric acid have been reported, there is prompt and striking reduction of plasma uric acid. Kuzell who treated 48 gouty patients with phenylbutazone reported that 40 were uniformly better on oral maintenance therapy with this drug than with previous conventional therapy.

The undesirable side effects include rash, edema, nausea, bloating, vertigo and sometimes typical ulcer symptoms. Nausea is the most frequent untoward reaction; pre-existing peptic ulcers may be activated and necessitate interruption of therapy. Experimental results in rats indicate that phenylbutazone apparently does not act on the pituitary-adrenal interrelationship as do cortisone and ACTH. (*See also* p. 239.)

Other Measures.—A large fluid intake is advisable and all alcoholic beverages should be avoided. If the urinary output falls too low, the high concentration of urates in the renal tubules leads to a precipitation of some of the urates as crystals, resulting in serious renal damage. Bartels[5] has used 20 per cent glucose intravenously in the treatment of acute attacks of gout.

During the [illegible]
morphine may [illegible]
longer than tw[illegible]

Regimen Between Attacks of Gout

Simplicity and moderation in eating. Foods containing large amounts of purine are forbidden. (*See* p. 767.)

[illegible]

each week, employed as a urate eliminant.

Benemid, 1 gram per day, is probably the most efficient uricosuric agent in interval gout with hyperuricemia.

[illegible]

Acute Episcleritis.—Ocular inflammations associated with gout are not numerous but such cases often present a perplexing problem. They are reported to impr

Chalk Gout.—

scripta and may

In circumscribed calcinosis, these concretions of calcium phosphate and calcium carbonate are confined almost exclusively to the toes, fingers, elbows and knees.[27,105] The subcutaneous calcium deposits may or may not be painful, aching and stiffness may be noted in nearby joints. Calcium metabolism reveals no abnormality, and no demineralization of bone or arthritic changes occur. The disease develops insidiously and is of unknown etiology and pathogenesis. (See also under Calcinosis in chapter on Collagen Diseases of the Scleroderma Group.)

DIAGNOSIS

The diagnosis of gouty arthritis in the initial or second attack is easy in the majority of cases. Usually it involves one joint in a male patient. It is the only arthritis which comes on with such unusual rapidity and com-

blood uric acid and a dramatic response to colchicine.

When a history of several acute attacks of joint pains with complete remission, particular

nosis is suggested.

in roentgenograms

increasing frequency, till eventually the joints are left with residual pain,

matic fever or rheumatoid arthritis, deposits of calcium, sebaceous cysts and ganglions.

Laboratory Tests.—A number of laboratory tests are useful in the diagnosis of gout. A determination of the blood or serum urate is of primary importance. This has been considered earlier. Any lesion suspected as a tophus should be needled or excised and examined for the presence of urates by microscope and the murexide test. X-ray examination is of

other evidence of decreased renal function may occur.[97,107] Anemia is seldom present in patients with gout except at times in the late stages of e disease.[42]

Premature resumption of normal activity may precipitate a flare-up. Strenuous physical therapy may provoke an exacerbation, a "cure-crisis."

Hench suggests that acute attacks of gout are better controlled when cinchophen (atophen) or a substitute for it are used to supplement other measures. He recommends giving 7½ grains (0.5 gm. of cinchophen)
4 times daily
amounts of c
in 4 cases in which colchicine had failed to relieve the pain and believes that
widespread fear
remedy. Others,
in acute gout

Intra-articular Injection of Hydrocortisone.—As a local adjunct in treatment of the acute attack of gouty arthritis, Hollander, *et al.*[154] have injected 25 mg. of hydrocortisone suspension directly into the inflamed joint. In 17 such cases, where a gouty bunion joint, ankle, wrist, or knee was so injected, there was a rapid defervescence of symptoms and signs within twelve hours in each instance. When this local measure was accompanied by the administration of colchicine, Butazolidin, or other general measures, no recurrence of the acute attack was noted during the subsequent period of observation.[155]

This local method produces a non-specific suppression of inflammation without effect on the cause. It is of use only in an occasional case, and *should never alone be regarded as treatment for the gouty attack.* The technic is described in the Chapter on Intra-articular Injection of Hydrocortisone.

INTERVAL TREATMENT

The general management of the patient with gout is definitely not completed when the acute attack is over. This is a chronic disease for which treatment must be continued indefinitely. The aim of treatment in the intervals between attacks of acute gouty arthritis is to avoid or postpone the number and severity of relapses. Unfortunately, the methods employed are not as satisfactory as those used in the treatment of acute gout; however, most writers favor some type of interval therapy. The usual regimen is planned to restrict the amount of purines and fat in the diet and increase the urate excretion by the periodic use of salicylates, cinchophen, or Benemid

Diet.—There is no altogether convincing evidence to indicate the efficiency of severe restriction of purine, protein or alcohol intake It is now *clear from studies using isotope technique that ingested preformed* purines are by no means the only source of uric acid since they are synthesized in the body from the simplest carbon derivatives of carbohydrate

-ines. The low
l and the high

The question of the value of the adherence to a rigorous purine-free diet in preventing recurrences of gouty arthritis has evoked considerable discussion in the past few years.

Laboratory Findings

Increased blood *uric acid* (*almost invariably present*).
Sedimentation rate usually normal between attacks, frequently elevated during attacks [illegible]
Roentgenograms generally negative early, of little use in diagnosis until late in the disease, roentgen-rays of the feet usually show signs of gout earlier than the hands. *Roentgen-rays show areas of erosion in only 52 per cent of cases*
Mild or moderate *leukocytosis* is the rule during attacks.
Anemia is seldom present in patients with gout except perhaps in the terminal stages [illegible]
Decrease in *renal function* may occur [illegible]

Main Symptoms and Signs of Gout

Tophi present in from 21 to 48 per cent of cases at any given time [illegible]
Elevation of *blood uric acid* almost invariably present
Acute *recurrent attacks with complete remissions* (before the development of secondary changes in joints).
Predominance in males (95 per cent of cases)
Involvement of big toe in initial attack in 70 per cent of cases, and sooner or later in 90 per cent
Roentgen-rays of little diagnostic value early in the disease, bony erosions seen in only about one-third of cases even in the later stages

Axioms of Hench, Suspect Gout If

Acute joint symptoms follow an *operative procedure*
There is a *family history* of gout.
There is a history of *olecranon bursitis* or Achilles tendinitis
Acute joint symptoms follow slight trauma, or excessive holiday food or drink.
Sudden joint symptoms appear during early morning hours
Acute arthritis occurs in men over the age of forty, in the absence of acute trauma or a history of gonorrhea.
[illegible] joint symptoms develop within minutes or a few hours
[illegible] symptoms are *associated with a chronic nephritis or renal colic*
[illegible] occur in patients with *polycythemia*, *leukemia* or after [illegible] the procedures such as injection of liver extract, salyr- [illegible] ergotamine tartrate (gynergen), thiamin chloride, decholin [illegible] acids or the use of a ketogenic diet
[illegible] or strenuous exercise precedes the first attack
[illegible] occur after hunting and fishing expeditions
[illegible] occur in the spring or fall.
[illegible] severe
[illegible] *of the great toe occurs*
[illegible] years wearing a shoe cut out over the metatarsophalangeal [illegible] great toe (sign of the split shoe)
[illegible] of an acute arthritis occurs within several weeks
[illegible] are *relieved* within twenty-four to seventy-two hours by ade- [illegible] of *colchicine*.
[illegible] absolutely *free from joint symptoms between attacks*
[illegible] fistulas or ulcers are present
[illegible] in a monarticular arthritis are entirely disproportionate to [illegible] findings
[illegible] reaction, especially the fever in any polyarthritis, is less than [illegible] findings might indicate [illegible]

Table 16. Low Purine Diet[14]

Purine Content of Foods per 100 Gm.		
List A (150-1000 mg)	List B (50-150 mg)	List C (0-15 mg)
Sweetbreads	Meats	Vegetables
Anchovies	Fish	Fruits
Sardines	Sea Food	Milk
Liver	Beans	Cheese
Kidney	Peas	Eggs
Meat Extractives	Lentils	Cereal
	Spinach	

SPECIAL INSTRUCTIONS

1. Avoid liver, sweetbreads, brains and kidneys A 2 oz portion of any other meat, fish or fowl may be served twice weekly.
2. Serve cheese and eggs as meat substitute Fish roe and caviar may be used as desired
3. Use 1 to 2 pints of milk daily in order to meet the protein need.
4. Omit all meat extractives, broth soups, and gravies
5. A moderate reduction should be made in the following vegetables: beans, peas, lentils and spinach
6. Allow fruits of all kinds—fresh, canned and dried
7. Allow cereals of all kinds
8. Serve sugar as desired with amount adjusted to caloric needs Cream and butter may be restricted when a low caloric allowance is needed or when a low fat regimen is desired

The approximate content of the above diet is 11 mg of purine nitrogen or 34 mg of uric acid

Since uric acid is derived from cell nuclei, the basic principle in the regulation of the purine content of a diet for gout must be the avoidance of those *foods in which nuclei are abundant* A reasonably low purine intake is assured by [illegible]
in table 16.
sea food, b [illegible]

most patients

The low fat intake presumably *prevents the retention of uric acid* Bartels has recently advocated a fat-free diet as a part of the regimen of the therapy of gout between attacks At the present time there is no agreement about the value of this diet, and according to one report the amount of fat in a well-balanced diet causes no significant retention of uric acid.[7] Furthermore, *in their experience the omission of fat from the diet has not* proved beneficial

Alcohol has been suggested as a precipitating cause of acute attacks of gout but the evidence is not conclusive. In view of the fact that, despite complete abstinence, many patients continue to suffer from repeated bouts

Acute Episcleritis.—Ocular inflammations associated with gout are not numerous but such cases often present a perplexing problem. They are reported to improve following the reduction of blood uric acid [73]

Chalk Gout.—This condition is more commonly called calcinosis circumscripta and may present swellings over joints which *simulate* gouty tophi. In circumscribed calcinosis, these concretions of calcium phosphate and calcium carbonate are confined almost exclusively to the toes, fingers, elbows and knees.[27,105] The subcutaneous calcium deposits may or may not be painful; aching and stiffness may be noted in nearby joints. Calcium metabolism reveals no abnormality, and no demineralization of bone or arthritic changes occur. The disease develops insidiously and is of unknown etiology and pathogenesis. (See also under Calcinosis in chapter on Collagen Diseases of the Scleroderma Group)

DIAGNOSIS

The diagnosis of gouty arthritis in the initial or second attack is easy in *the majority of cases. Usually it involves one joint in a male patient* It is the only arthritis which comes on with such unusual rapidity and commonly after some characteristic provocative, such as minor trauma, dietary indiscretion, surgical operations or strenuous exercise. The excruciating pain is follow [illegible] ing tissues. [illegible] *no residual* [illegible] blood uric acid and a dramatic response to colchicine.

When a history of several acute attacks of joint pains with complete remission, particularly in an obese middle-aged man, is obtained, the diagnosis is suggested. The presence of subcutaneous tophi and areas of erosion in roentgenograms are diagnostic features. Attacks tend to occur with increasing frequency, till eventually the joints are left with residual pain, stiffness and deformity. Olecranon bursitis is frequent with gout and should raise the suspicion of this disease. [illegible]

and ganglions.

Laboratory Tests.—A number of laboratory tests are useful in the diagnosis of gout. A determination of the blood or serum urate is of primary importance. This has been considered earlier. Any lesion suspected as a tophus should be needled or excised and examined for the presence of urates by microscope and the murexide test. X-ray examination is of value in the diagnosis of the late stages of gout. The changes most suggestive of gout are well-defined, punched-out areas located in the subchondral [illegible] of the base or head of the phalanges. [illegible]

other evidence [illegible] seldom present in patients with gout except at times in the late stages of the disease.[42]

of joint pain and teetotalers develop classical gout, some clinicians permit use of alcoholic beverages.[54] Complete avoidance of the use of alcohol is advised by others.[6,56,63] We prefer abstinence from alcoholic beverages.

It has been supposed that coffee, tea and chocolate give rise to uric acid and so increase tophaceous deposits. It has been shown,[136] that the methylxanthines (caffeine, theophylline, and theobromine) of these beverages are metabolized to methyl-urates; these substances are chromogens in the uric acid tests, but are not deposited in the gouty tophus. It appears that since only true uric acid is deposited in the tophus, there may be little reason to eliminate these items from the diet.

Drugs.—Talbott believes that most gouty patients should take some colchicine from time to time between attacks of gout. In those individuals who have less than one attack of gout per year, a few colchicine tablets ($\frac{1}{2}$ mg.) may be taken at times when mild pains and aches appear in the joints.[118] Patients who recognize prodomata of an attack may abort attacks by prompt use of colchicine. If these premonitory symptoms of a gouty attack do not materialize in a few hours, further ingestion of colchicine is unnecessary. In some patients from 1 to 2 tablets per week up to 1 or more per day are taken. In a few patients with severe gouty arthritis, 2 or 3 tablets of colchicine have been given daily for five years with no untoward effects from the prolonged ingestion of the drug. The individual does not develop tolerance to the drug and the full course of colchicine is still just as effective in these individuals should an acute attack occur. There is increasing evidence that colchicine has a pre-eminent place in the prophylaxis of acute gout where there is frequent recurrence of attacks.

[illegible]

of Benemid, cinchophen and sodium salicylate were the two drugs most commonly employed, acetylsalicylic acid and neocinchophen were also used. Since Benemid appears to be a relatively safe drug, it is the most satisfactory uricosuric agent available. It should be emphasized that Benemid has no analgesic action and is of no value in the treatment of the acute gouty attack.

Benemid.—A number of clinical studies[152,163,170] indicate that Benemid is an effective uricosuric drug and of definite benefit in chronic gouty arthritis. There is general agreement that the incidence of acute attacks of gout and the severity of attacks diminishes with continued therapy. The most generally useful dose is 1.0 gram per day. Several patients maintained on protracted Benemid medication have noted disappearance or decrease in size of tophi together with lessened swelling of chronically enlarged joints.[181] In a few patients acute attacks of gout have developed soon after. Much more experience is required before we can make a final evaluation of this drug; at present it appears to be the most satisfactory uricosuric agent available.

Cinchophen.—Cinchophen (atophen) is used routinely by some in the interval treatment of gout except for those patients with hepatic dysfunction or an idiosyncrasy to cinchophen. The plan of Graham[46] is advocated [illegible] consecutive [illegible] e blood uric [illegible] omitted or

Laboratory Findings

Increased blood *uric acid* (*almost invariably present*).
Sedimentation rate usually normal between attacks; frequently elevated during attacks [12]
Roentgenograms generally negative early; of little use in diagnosis until late in the disease, roentgen-rays of the feet usually show signs of gout earlier than the hands *Roentgen-rays show areas of erosion in only 52 per cent of cases*
Mild or moderate *leukocytosis* is the rule during attacks
Anemia is seldom present in patients with gout except perhaps in the terminal stages [81]
Decrease in *renal function* may occur.[25,107]

Main Symptoms and Signs of Gout

Tophi present in from 21 to 48 per cent of cases at any given time [12]
Elevation of *blood uric acid* almost invariably present.
Acute *recurrent attacks with complete remissions* (before the development of secondary changes in joints)
Predominance in males (95 per cent of cases)
Involvement of big toe in initial attack in 70 per cent of cases, and sooner or later in 90 per cent.
Roentgen-rays of *little diagnostic value early* in the disease, bony erosions seen in only about one-third of cases even in the later stages

Axioms of Hench; Suspect Gout If

Acute joint symptoms follow an *operative procedure*.
There is a *family history* of gout
There is a history of *olecranon bursitis* or Achilles tendinitis
Acute joint symptoms follow slight trauma, or excessive holiday food or drink
Sudden joint symptoms appear during early morning hours
Acute arthritis occurs in men over the age of forty, in the absence of acute trauma or a history of gonorrhea
Acute joint symptoms develop within minutes or a few hours
Joint symptoms are *associated with a chronic nephritis or renal colic*
Joint symptoms occur *in* patients with *polycythemia*, *leukemia* or after certain therapeutic procedures such as injection of liver extract, salyrgan, insulin, ergotamine tartrate (gynergen), thiamin chloride, decholin (dehydrocholic acid) or the use of a ketogenic diet
A long automobile ride or strenuous exercise precedes the first attack
Joint symptoms occur after hunting and fishing expeditions
Joint symptoms recur in the spring or fall
Joint pain is exceptionally severe
Acute *involvement of the great toe occurs*
The patient appears wearing a shoe cut out over the metatarsophalangeal joint of the great toe (sign of the split shoe)
Complete recovery of an acute arthritis occurs within several weeks
Symptoms are *relieved* within twenty-four to seventy-two hours by adequate doses of *colchicine*.
The patient is absolutely *free from joint symptoms between attacks*
Periarticular fistulas or ulcers are present.
The symptoms in a monarticular arthritis are entirely disproportionate to the findings
The general reaction, especially the fever in any polyarthritis, is less than the findings might indicate [124,125]

given for a while only one or two days a week in the usual or in smaller doses three days a week. These patients are advised as to the symptoms of cinchophen toxicity (anorexia, dyspepsia, nausea, pruritis, jaundice) and

drug is never employed in some clinics Those who advocate or condone its use are willing to assume this small risk if gout is incontrollable otherwise. This is done in an effort to prevent the articular, renal and vascular complications of gout.

Hueper[44] in a critical review of the literature of cinchophen, advises every

phen is justified

risk inherent in the latter drug. Because this reduction of the blood urate level is temporary (about three months), the program of large doses of salicylates is recommended only for patients with rapidly recurring gouty attacks. To control the gastrointestinal symptoms following massive doses of salicylates equal amounts of sodium bicarbonate are prescribed.

Glycine and Salicylates —The synergistic action between salicylates and

thiamine

Treatment of Chronic Gouty Arthritis.—The results of therapy in this

ts are permanently

A few physicians

helpful. The long-term use of uricosuric agents in chronic gouty arthritis, with or without tophi, is recommended Benemid appears to be the most satisfactory drug so far available. Large doses of salicylates are often necessary to control pain Fever therapy and typhoid vaccine reactions have been used occasionally *Hydrotherapy, massage and exercises must be used cautiously*

Roentgen Examination.—Huber in 1896 first described the specific "punched-out" areas of bone seen roentgenologically in gout.[65] These *areas represent osseous tophi containing uric acid crystals and although* they may be found in any bone, involvement of the lower extremities (Fig. 237) is more frequent than the upper.[18,88,103] Roentgenograms are frequently negative early in the disease; bony erosions are seen in only about one-third of cases even in the later stages. Joints rarely involved by gout include the sacro-iliac, temperomandibular, sternoclavicular articulations, spine, shoulders or hips.[90] Recent autopsy material has shown gouty erosions of the sacro-iliac joints and urate deposits in the spine and jaw.[7]

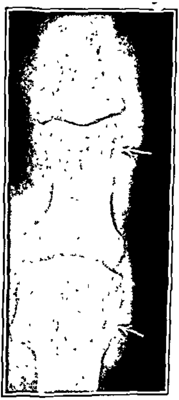


FIG. 240 —Punched-out areas in a sixty-two year old brewmaster with gout This great toe had suffered repeated attacks of acute swelling

The most suggestive roentgen findings in gout are punched-out (cystic) areas which are usually 5 mm. or more in diameter, located in the subchondral bone of the base or head of the phalanges in the hands or feet. [illegible] stage of

hypertrophy or spurring is also common in joints [illegible] seat of numerous attacks of gout. Occasionally, decalcification occurs in patients with prolonged severe attacks Localized soft tissue tumefactions, with or without calcifications, may be observed.

of joint pain and teetotalers develop classical gout, some clinicians permit use of alcoholic beverages.[54] Complete avoidance of the use of alcohol is advised by others.[6, 56, 63] We prefer abstinence from alcoholic beverages.

It has been supposed that coffee, tea and chocolate give rise to uric acid and so increase tophaceous deposits. It has been shown,[136] that the [illegible]

[illegible] be little

[illegible] take some colchicine from time to time between attacks of gout. In those individuals who have less than one attack of gout per year, a few colchicine tablets ($\frac{1}{2}$ mg.) may be taken at times when mild pains and aches appear in the joints.[118] Patients who recognize prodomata of an attack may abort attacks by prompt use of colchicine If these premonitory symptoms of a gouty attack do not materialize in a few hours, further ingestion of colchicine is unnecessary. In some patients from 1 to 2 tablets per week up to 1 or more per day are taken In a few patients with severe gouty arthritis, 2 or 3 tablets of colchicine have been given daily for five years with no untoward effects from the prolonged ingestion of the drug. The individual does not develop tolerance to the drug and the full course of colchicine is still just as effective in these individuals should an acute attack occur. There is increasing evidence that colchicine has a pre-eminent place in the prophylaxis of acute gout where there is frequent recurrence of attacks

Urate Diuretics.—Certain agents produce an increase in the excretion of uric acid and a decrease in the blood uric acid level in gouty or in normal subjects and have been called the *uricosuric drugs*. Until the introduction of Benemid, cinchophen and sodium salicylate were the two drugs most commonly employed, acetylsalicylic acid and neocinchophen were also used Since Benemid appears to be a relatively safe drug, it is the most satisfactory uricosuric agent available It should be emphasized that Benemid has no analgesic action and is of no value in the treatment of the acute gouty attack

Benemid.—A number of clinical studies[152, 163, 170] indicate that Benemid is an effective uricosuric drug and of definite benefit in chronic gouty arthritis. There is general agreement that the incidence of acute attacks of gout and the severity of attacks diminishes with continued therapy. The most generally useful dose is 1.0 gram per day. Several patients maintained on protracted Benemid medication have noted disappearance or decrease in size of tophi together with lessened swelling of chronically enlarged joints.[181] In a few patients acute attacks of gout have developed soon after. Much more experience is required before we can make a final evaluation of this drug, at present it appears to be the most satisfactory uricosuric agent available.

Cinchophen.—Cinchophen (atophen) is used routinely by some in the interval treatment of gout except for those patients with hepatic dysfunction or an idiosyncrasy to cinchophen. The plan of Graham[46] is advocated by many: cinchophen $7\frac{1}{2}$ grains (0 5 gm.) 3 times daily for three consecutive days each week. The drug is continued thus indefinitely; if the blood uric acid becomes normal or nearly so, the drug may be temporarily omitted or

areas in bones which are matoid arthritis, degenera- roidism, multiple myeloma, umma, tuberculosis, yaws, metastatic malignancy and leprosy may occasionally present roentgen findings similar to those of gout.

Although osseous tophi may be found in almost any bone, involvement in the lower extremities is more frequent than the upper [18, 88, 108] In suspected cases, the feet (and particularly the great toes) should always be examined for such lesions

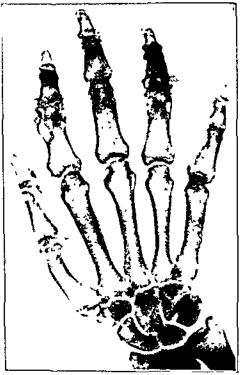


Fig 211 —Gout Note punched-out areas in several bones

PROVOCATIVE FACTORS

A number of factors play some part in determining individual attacks of gouty arthritis and its site, but their true relation to the disease has not been determined

Trauma.—Trauma has repeatedly been considered responsible as a provocative agent for acute attacks of gout. The first metatarsophalangeal joint is liable to trauma and chronic strain from pressure of tight shoes. Attacks may result from such minor trauma as that from long walks, incident to hunting or fishing trips, sight-seeing or golf.

given for a while only one or two days a week in the usual or in smaller doses three days a week. These patients are advised as to the symptoms of cinchophen toxicity (anorexia, dyspepsia, nausea, pruritis, jaundice) and

Salicylates.—Recent well-controlled studies[72,103] indicate that salicylates in continuous daily doses of 5 to 6 gm. are effective in promoting the excretion of uric acid and effecting a lowering of the blood urate level. The results obtained were equal to those ascribed to cinchophen without the risk inherent in the latter drug. Because this reduction of the blood urate level is temporary (about three months), the program of large doses of salicylates is recommended only for patients with rapidly recurring gouty at

do

author suggests that an attempt be made to show that glycine really does

thiamine

Treatment of Chronic Gouty Arthritis.—The results of therapy in this

ermanently

physicians

The long-

term use of

tophi, is rec

so far availa

pain Fever

casionally. *Hydrotherapy, massage and exercises must be used cautiously*

Dietary Excesses.—Gouty arthritis is often provoked by dietary indiscretions associated with feast days, holidays, weddings. Bauer[9] cautions against accepting as verified the influence of dietary habits on the development and progress of gout.

Lockie[79] has suggested that in questionable cases, a diet high in fat (300 gm.) and low in protein (50 gm.) and carbohydrate (25 gm.) be administered; if this is followed within a few days by an exacerbation of joint symptoms, gout should be suspected. This diet should be continued for a period of from five to seven days as a therapeutic test. McEwen[87] used a high fat diet and each of five gouty persons had acute attacks. Some consider these tests unreliable.

Drugs.—Medicinal provocatives include liver extracts,[33,34,35,94,117] gold,[19] thiamin chloride,[131] sulfathiazole, dehydrocholic acid,[77] insulin,[58] salyrgan,[95] gynergen, and adrenocorticotropin (ACTH).[52]

Alcohol.—An association of gout
noted since gout has been known
excesses in drink the most import
the clinical course of gout is characterized by sudden unexplained changes in patients with gout who never ingest alcohol, it is difficult to evaluate the rôle of excesses in drink in precipitating acute gout.

Surgical Operation.—Surgical operations are prone to provoke attacks
ce of
era-
tions. Acute arthritis developed within the third to fifth postoperative day in 5 cases of gout reported by Ficarra and Adams.[36] Whereas rheumatoid arthritis is often temporarily benefited by any operative procedure, the reverse is true in gout.

Miscellaneous Factors.—Latent gout has been activated by marked blood loss and transfusions, chronic glomerular nephritis, leukemia, polycythemia,[100] as well as by emotional upsets, foreign protein therapy, exposure to cold and dampness and severe purgation.

TREATMENT OF GOUT

Although there is no known cure for gout, the control of the frequency and severity of acute attacks is quite satisfactory with the measures avail-
derately and
re, excessive
trauma to joints, excessive foods high in purines,
ovocatives of acute gouty arthritis.
The patient should be at absolute bed rest, the affected

lest another attack be provoked. There is no specific treatment for any of the later complications of gout.

Treatment of Tophi.—*If* tophi on the hands or feet become large or ulcerate, excision with thorough débridement results in prompt healing and obviates the dangers of infection and possibly amputation.[36] Linton and Talbott reported on the surgical treatment of tophaceous gout based on such therapy in 11 patients.[78] Surgical treatment of gouty tophi is indicated when large deposits have developed and must be removed for cosmetic reasons or because pain, interference with movements, discharging sinus, or extensive phalangeal involvement of the toes and fingers. Follow-up studies showed that only 3 of 93 tophi removed showed recurrences of any size. In all patients in whom tophi were removed because they were causing pain, the results were satisfactory. Surgery should be done early in the course of tophi if these are causing symptoms.

PROGNOSIS

Prognosis is poorest in those whose symptoms appear early in life and best in those whose symptoms come on late in life. Gout is a chronic disorder which is not cured when the acute attack has subsided. Treatment must be continued for life in order to prevent recurrence of symptoms. Faithful adherence to treatment (especially between acute attacks) will modify the course, reduce the severity and number of attacks, reduce the likelihood or prevent the occurrence of chronic gouty arthritis, and possibly postpone or avoid fatal renal and cardiovascular complications.

BIBLIOGRAPHY

[illegible] Jour, 10, 551, 1916
[illegible], H · Arch Int Med, 70, 101, 1942
[illegible] Pub Health and Hygiene, 1, 298, 1938
[illegible] Jour Biol. Chem, 151, 123, 1943
[illegible] Ann Int Med, [illegible], 1943
[illegible] eases of Metabolism, 2d
[illegible] 2, 161, 1931
[illegible] 35
[illegible] Leipsic, F. C. W. Vogel, 1927.
19. Brugsch, T. Z ges exp Path Therap, 2, 619, 1905
20. Buchanan, O H., Block, W D, and Christman, A. A.: Jour. Biol Chem, 157, 181, 1945.
21. Bulger, H. A, and Johns, H. E: Jour. Biol. Chem, 140, 427, 1940
22. Bunim, J J, and McEwen, C.: Arch Path, 29, 700, 1940.
23. Claiborne, T S Jour Am. Med Assn., 115, 38, 1940
24. Cmunt, E. Arch Med. Hydrol, 14, 146, 1936
25. Cohen, A.. South Med and Surg, 98, 637, 1936.
26. ———: *Ibid*, 103, 654, 1941.

27 COMROE, B I, CHAMBERLIN, G, and SUNDERMAN, W. F Am. Jour Roentgenol, 41, 749, 1939

W. V., and TALBOTT,

sn, 110, 2149, 1938

Biol Chem, 54, 595, 1922

Copenhagen, 102, 1157, 1940

39 FOLIN, O, and DENIS, W. Arch Int Med, 16, 33, 1915
40 FOLIN, O, and LYMAN, H Jour. Pharmacol and Exper Therap, 4, 539, 1912–1913
41 GRABFIELD, G P: Transactions of the Association of American Physicians, 51, 331, 1936
42 ——— Ann Int. Med, 11, 651, 1937.
43 GRABFIELD, G P, and KNAPP, E: Jour Pharmacol and Exper. Therapy, 32, 341, 1928
44 GRABFIELD, G P, PRESCOTT, B, and SWAN, W K. Jour Pharmacol and Exper. Therapy, 61, 293, 1937
45 GRABFIELD, G P, and PRATT, J H Jour. Pharmacol and Exper Therapy, 42, 407, 1931
46 GRAHAM, G Proc Roy Soc. Med, 20, 1, 1927
47 GUDZENT, F
48 ———
49 HADEN, R
50 ———
51 ———
52 HELLMAN, L Science, 109, 280, 1949.
53 HENCH, P S Jour Am Med Assn, 116, 453, 1941.
54 HENCH, P S, *et al.* Ann. Int Med, 28, 66, 1948
55 HENCH, P S Proc Staff Meet Mayo Clin, 11, 476, 1936
56 ——— Jour Am Med Assn, 116, 453, 1941
57 HENCH, P S, and DARNALL, C M Med Clin North America, 16, 1371, 1933
58 HENCH, P S Nelson's Loose Leaf Living Surgery, New York, Thomas Nelson & Sons, 3, 104, 1935

PART VIII

ARTHRITIS CAUSED BY OR OCCURRING WITH SPECIFIC INFECTIONS

CHAPTER 43

GONORRHEAL ARTHRITIS

By Victor G. Balboni, M.D.

The incidence of gonorrheal arthritis has decreased markedly since the advent of sulfonamides and penicillin. In the presulfonamide era arthritis complicated 1 to 5 per cent of cases of gonorrheal urethritis. However, with present da
0.3 per cent.[42]
has also been
therapy

In view of the devastating nature of the disease if untreated (the marked destruction of cartilage and bone), and because of the highly effective therapy of this disorder (penicillin), it is important that every effort be made to diagnose it in the earliest stage.[33] *Treatment of gonorrheal arthritis is now a fairly simplified procedure, diagnosis is often faulty.* Typical gonorrheal arthritis is a metastatic infection of the articular tissue by the gonococcus. It must be differentiated from rheumatoid arthritis precipitated by a gonococcal infection of the urethra or prostate.

DIAGNOSTIC FACTORS

1. History
2. Physical examination
3. Laboratory tests (urethral smears and cultures for gonococci, smears and cultures of synovial fluid for gonococci, leukocyte count on synovial

cent, (2) *ankle*—38 per cent, (3) *metatarsophalangeal joints*—28 per cent, (4) *wrists*—23 per cent, (5) *fingers*—22 per cent, (6) *shoulder*—20 per cent, (7) *metacarpophalangeal joints*—17 per cent, (8) *toes*—14 per cent, (9) *other joints*: hip—10 per cent, elbow—8 per cent, sacro-iliac—6 per cent, intervertebral—5 per cent, heel—5 per cent, temporomandibular—2 per cent, sternoclavicular—2 per cent, olecranon bursa—0.7 per cent, acromioclavicular—0.7 per cent.[61,64]

lest another attack be provoked. There is no specific treatment for any of the later complications of gout.

Treatment of Tophi.—If tophi on the hands or feet become large or ulcerate, excision with thorough débridement results in prompt healing and obviates the dangers of infection and possibly amputation.[56] Linton and Talbott reported on the surgical treatment of tophaceous gout based on such therapy in 11 patients.[78] Surgical treatment of gouty tophi is indicated when large deposits have developed and must be removed for cosmetic reasons or because pain, interference with movements, discharging sinus, or extensive phalangeal involvement of the toes and fingers. *Follow-up studies showed that only 3 of 93 tophi removed showed recurrences of any size. In all patients in whom tophi were removed because they were causing pain, the results were satisfactory.* Surgery should be done early in the course of tophi if these are causing symptoms.

PROGNOSIS

Prognosis is poorest in those whose symptoms appear early in life and best in those whose symptoms come on late in life. Gout is a chronic disorder which is not cured when the acute attack has subsided. Treatment must be continued for life in order to prevent recurrence of symptoms. Faithful adherence to treatment (especially between acute attacks) will modify the course, reduce the severity and number of attacks, reduce the likelihood or prevent the occurrence of chronic gouty arthritis, and possibly postpone or avoid fatal renal and cardiovascular complications.

BIBLIOGRAPHY

1. [illegible]
2. [illegible]
3. [illegible]
4. [illegible]
5. BARTELS, E C. Ann Int Med, 18, 21, 1943
6. [illegible]
7. [illegible]
8. [illegible] bolism, 2d
9. [illegible]
10. [illegible]
11. [illegible]
12. [illegible]
13. [illegible]
14. [illegible]
15. [illegible]
16. [illegible]
17. [illegible] W Vogel,
18. [illegible] 1927.
19. BRUGSCH, T Z ges exp Path. Therap, 2, 619, 1905
20. BUCHANAN, O H., BLOCK, W. D, and CHRISTMAN, A A Jour Biol Chem, 157 181, 1945
21. BULGER, H A, and JOHNS, H E: Jour Biol Chem, 140, 427, 1940
22. BUNIM, J J, and MCEWEN, C.: Arch Path, 29, 700, 1940
23. CLAIBORNE, T S Jour Am. Med Assn, 115, 38, 1940
24. CMUNT, E. Arch Med Hydrol, 14, 146, 1936.
25. COHEN, A.: South Med. and Surg, 98, 637, 1936
26. ———: *Ibid*, 103, 654, 1941.

BIBLIOGRAPHY

1. Abbott, L D, Jr, Mandeville, F. B., and Rein, W. J: Virginia Med Monthly, 70, 615-617, 1943.
2 Albrecht, H: Ztschr f Heilk, 23, 366, 1902
3 Bodecker, C.. Ztschr f rat. Med, 7, 130, 1859
4. ———· Ann d. Chem u Pharmakol, 117, 98, 1861
5 Brune, T. B · Boston Med and Surg Jour, 115, 621, 1886
5a Cope, C. B, and Kassandfr, P. Jour. Am. Med. Assn, 150, 997, 1952
6 Crosfill, J W. L. Jour Roy Nav Med Serv., 31, 173-175, 1945
7. Cushing, E H., and Stout, A P · Arch Surg, 12, 539, 1926
8 Fishberg, E H · Jour Am. Med Assn., 119, 882, 1942
9 Garrod, A B · Inborn Errors of Metabolism, 2nd. ed, London, Oxford Univ. Press, 1923
10. Gould, J N, and Thomas, T. G · Brit Med. Jour, 1, 788, 1943
11. Hench, P S, et al Ann Int. Med. 28, 310, 1948
12. Hertzberg, J Acta radiol, 26, 484-490, 1945.
13 Howard, C P, and Mills, E. S · Ochronosis, In Christian, H. A.· Oxford Medicine, 1941, Oxford University Press, New York, vol 4, pt 2, chap 9, pp 223-228
14 Leslie, A Arch Int Med, 71, 68-77, 1943
15 Pomerantz, M M., Friedman, L. J, and Tunick, I. S.: Radiology, 37, 295-302, 1941
16 Reed, J, and Sosman, M C · Radiology, 38, 579, 1942
17. Reiss, O, and Kato Am. Jour. Dis Child, 43, 365, 1932
18 Schein, A J, and Arkin, A M.· Jour. Bone and Jour. Surg, 24, 396, 1942
19 Sealock, R R, Gladston, M and Steele, J. M. Proc. Soc Exper. Biol and Med, 44, 580, 1940
20 Smith, J. W Jour. Am. Med. Assoc, 120, 1282-1288, 1942.
21. Soderbergh, G · Neurol Centralbl, 21, 1362, 1913.
22 Swirsky, M Y Clinics, 2, 1323, 1333, 1944
23. Virchow, R. Virchows Arch f path Anat, 37, 212, 1866
24 Welt, S, Rosenthal, N, and Openheimer, B. S: Jour. Am. Med Assn, 92, 637, 1929
25 Young, H H.· Jour Urol, 41, 48-58, 1944

27. Comroe, B I, Chamberlin, G, and Sunderman, W. F Am Jour. Roentgenol, 41, 749, 1939
28 Coombs, F. S, *et al* Jour. Clin Invest., 19, 525, 1940
29. Coombs, F. S, Pecora, L. J, Thorogood, E, Consolazio, W V, and Talbott, J. H · Jour Clin Invest, 19, 525, 1940
30 Christopher, F, and Monroe, S E: Jour Am Med. Assn, 110, 2140, 1938
31. Davis, A. R, Newton, E B, and Benedict, S. R · Jour. Biol. Chem, 54, 595, 1922

Copenhagen, 102, 1157, 1940
36 Ficarra, B J., and Adams, R · Arch Surg, 1, 229, 1945

1913
41. Grabfield, G P.: Transactions of the Association of American Physicians, 51, 331, 1936.
42 ——— Ann Int Med, 11, 651, 1937.
43 Grabfield, G P, and Knapp, E Jour Pharmacol and Exper. Therapy, 32, 341, 1928
44 Grabfield, G P, Prescott, B, and Swan, W K Jour. Pharmacol. and Exper Therapy, 61, 293, 1937
45 Grabfield, G P, and Pratt, J H. Jour Pharmacol and Exper Therapy, 42, 407, 1931
46 Graham, G Proc Roy Soc Med, 20, 1, 1927
47 Gudzent, F. Ein Lehrbuch f Arzte Studierende, Berlin, Julius Springer, 1928.
48 ——— Gicht und Rheumatismus, Berlin, Julius Springer, 1928
49 Haden, R L Cleveland Clin. Quart, 8, 230, 1941
50 ——— Proc Interstate Postgrad Med Assn North America, 259, 1941
51 ——— Clinics, 1, 531, 1942
52 Hellman, L Science, 109, 280, 1949
53 Hench, P S Jour Am Med Assn., 116, 453, 1941
54 Hench, P S, *et al* Ann Int Med, 28, 66, 1948
55 Hench, P S Proc Staff Meet Mayo Clin, 11, 476, 1936
56 ——— Jour Am Med. Assn, 116, 453, 1941
57 Hench, P S, and Darnall, C M Med Clin North America, 16, 1371, 1933
58 Hench, P S Nelson's Loose Leaf Living Surgery, New York, Thomas Nelson & Sons, 3, 104, 1935

Am Phys, 43, 217, 1928

, 192, 483, 1936

Jour Bone and Joint

, 24, 429, 1940

3, 950, 1944

PART VIII

ARTHRITIS CAUSED BY OR OCCURRING WITH SPECIFIC INFECTIONS

CHAPTER 43

GONORRHEAL ARTHRITIS

By Victor G Balboni, M.D

The incidence of gonorrheal arthritis has decreased markedly since the advent of sulfonamides and penicillin In the presulfonamide era arthritis complicated 1 to 5 per cent of cases of gonorrheal urethritis However, with present

0 3 per cent [4]

has also bee

therapy

gonorrheal arthritis is a metastatic infection of the articular tissue by the gonococcus It must be differentiated from rheumatoid arthritis precipitated by a gonococcal infection of the urethra or prostate

DIAGNOSTIC FACTORS

1 History
2 Physical examination

cent, (2) *ankle*—38 per cent, (3) *metatarsophalangeal joints*—28 per cent, (4) *wrists*—23 per cent, (5) *fingers*—22 per cent, (6) *shoulder*—20 per cent, (7) *metacarpophalangeal joints*—17 per cent, (8) *toes*—14 per cent; (9) *other joints* hip—10 per cent, elbow—8 per cent, sacro-iliac—6 per cent, intervertebral—5 per cent, heel—5 per cent, temporomandibular—2 per cent, sternoclavicular—2 per cent, olecranon bursa—0 7 per cent, acromioclavicular—0.7 per cent.[51, 66]

lest another attack be provoked. There is no specific treatment for any of the later complications of gout.

Treatment of Tophi.—If tophi on the hands or feet become large or ulcerate, excision with thorough débridement results in prompt healing and obviates the dangers of infection and possibly amputation.[55] Linton and Talbott reported on the surgical treatment of tophaceous gout based on such therapy in 11 patients [78] Surgical treatment of gouty tophi is indicated when large deposits have developed and must be removed for cosmetic reasons or because pain, interference with movements, discharging sinus, or extensive phalangeal involvement of the toes and fingers. Follow-up studies showed that only 3 of 93 tophi removed showed recurrences of any [illegible]

PROGNOSIS

Prognosis is poorest in those whose symptoms appear early in life and best in those whose symptoms come on late in life. Gout is a chronic disorder which is not cured when the acute attack has subsided. Treatment must be continued for life in order to prevent recurrence of symptoms. Faithful adherence to treatment (especially between acute attacks) will modify the course, reduce the severity and number of attacks, reduce the likelihood or prevent the occurrence of chronic gouty arthritis, and possibly postpone or avoid fatal renal and cardiovascular complications.

BIBLIOGRAPHY

1 [illegible] 10, 551, 1916
2 [illegible] 1, 1942
3 [illegible] 8, 1938
4. [illegible] 1943
5. [illegible]
6 [illegible] 9, 86, 1917
7. [illegible]
8 [illegible] Metabolism, 2d
9. [illegible]
10 [illegible] 31
11 [illegible]
12 [illegible]
13 [illegible]
14 [illegible] Acta med Scandinav., 86, 235, 1935
15. [illegible]
16 [illegible]
17. [illegible] C W Vogel,
18. [illegible] 1927
19 BRUGSCH, T. Z ges exp. Path. Therap, 2, 619, 1905
20 BUCHANAN, O H, BLOCK, W. D, and CHRISTMAN, A. A.: Jour. Biol Chem, 157 181, 1945
21 [illegible] Jour. Biol. Chem, 140, 427, 1940.
22 [illegible], 29, 700, 1940
23 [illegible] 5, 38, 1940
24 CMUNT, L: Arch Med [illegible] 36.
25 COHEN, A.: South Med. and Surg, 98, 637, 1936
26 ——— *Ibid.*, 103, 654, 1941.

BIBLIOGRAPHY

1. Abbott, L D, Jr, Mandeville, F. B, and Rein, W J: Virginia Med Monthly, 70, 615–617, 1943
2 Albrecht, H: Ztschr f Heilk, 23, 366, 1902.
3 Bodecker, C.: Ztschr f. rat Med, 7, 130, 1859
4 ———: Ann d. Chem. u. Pharmakol, 117, 98, 1861.
5 Brune, T B: Boston Med. and Surg Jour, 115, 621, 1886
5a Cope, C B, and Kassander, P.. Jour Am. Med. Assn, 150, 997, 1952
6 Crosfill, J W. L. Jour. Roy Nav. Med Serv, 31, 173-175, 1945.
7 Cushing, E H, and Stout, A P.. Arch Surg, 12, 539, 1926
8. Fishberg, E H Jour Am Med Assn, 119, 882, 1942
9 Garrod, A B Inborn Errors of Metabolism, 2nd. ed, London, Oxford Univ. Press, 1923
10 Gould, J N, and Thomas, T. G: Brit Med Jour, 1, 788, 1943
11. Hench, P S, et al Ann Int Med 28, 310, 1948
12 Hertzberg, J: Acta radiol, 26, 484–490, 1945
13 Howard, C P, and Mills, E S.: Ochronosis, In Christian, H. A.: Oxford Medicine, 1941, Oxford University Press, New York, vol. 4, pt. 2, chap 9, pp 223–228
14. Leslie, A. Arch. Int Med, 71, 68-77, 1943
15 Pomerantz, M M, Friedman, L J, and Tunick, I S Radiology, 37, 295–302, 1941
16 Reed, J, and Sosman, M C. Radiology, 38, 579, 1942
17. Reiss, O, and Kato: Am Jour. Dis Child, 43, 365, 1932.
18. Schein, A. J, and Arkin, A. M. Jour Bone and Jour Surg, 24, 396, 1942
19 Sealock, R R, Gladston, M and Steele, J. M: Proc. Soc Exper. Biol and Med, 44, 580, 1940
20 Smith, J W: Jour Am Med Assoc, 120, 1282-1288, 1942
21 Soderbergh, G. Neurol Centralbl, 21, 1362, 1913
22 Swirsky, M Y: Clinics, 2, 1323, 1333, 1944
23 Virchow, R Virchows Arch f path Anat, 37, 212, 1866.
24 Welt, S, Rosenthal, N., and Oppenheimer, B S: Jour. Am. Med. Assn, 92, 637, 1929
25 Young, H H: Jour Urol, 41, 48–58, 1944.

124. THANNHAUSER, S J · Lehrbuch des Stoffwechsels und der Stoffwechsel-Krankheiten, München, J F /Bergmann, 1929
125. THOMSON, F. G · Brit. Jour Rheumat , 1, 25, 1938.
126 TURNER, D · Handbook of Diet Therapy, Chicago, University Chicago Press. p 41, 1946
127 [illegible]
128
129
130
131
132
133
134
135
136
1947
137 WOLFSON, W Q , COHN, C , LEVINE, R , and HUDDLESTUN, B Am. Jour. Med , 4, 774, 1948
[illegible]
141 BENEDICT, J D , FORSHAM, P H , ROCHE, M , SOLOWAY, S , and STETTEN, D JR.: Jour Clin Invest , 29, 1104, 1950
142 BENEDICT, J D , FORSHAM, P H , STETTEN, D JR. Jour Biol Chem , 181, 183, 1949
143 BERLINER, R. W , HILTON, J G , YÜ, T F , KENNEDY, T J JR Jour Clin Invest , 29, 396, 1950
144 BISHOP, C , GARNER, W , TALBOTT, J H Jr Clin. Invest , 30, 879, 1951
145 BISHOP, C Fed Proc , 9, 151, 1950
146 BOGER, W P , BEATTY, J O , PITTS, F W , FLIPPIN, H F Ann Int Med , 33, 18, 1950
147 BROCHNER-MORTENSEN, K Acta med Scandinav , 106, 81, 1941
148 BROWN, J , MALLORY, G K New England Jour Med , 243, 325, 1950
149 LAPEN, K Malayan Med Jour , 11, 117, 1836
150 FRIEDLANDER, R. D Jour Am Med Assn , 145, 11, 1951
151 FRIEDMAN, M , and BYERS, S O Am Jour. Med , 9, 31, 1950
152 GUTMAN, A B , and YÜ, T F Am. Jour Med , 9, 24, 1950
153 HOLLANDER, J L , BROWN, E M , ELKINTON, J R Proc Second Clin ACTH Conference, Philadelphia, Blakiston Company, 1951
154 HOLLANDER, J L , BROWN, E M , JESSAR, R. A , and BROWN, C Y Jour Am Med Assn , 147, 1629, 1951
155 HOLLANDER, J L Personal Communication
156 KIDD, E C , BOYCE, K C , and FREYBERG, R H Am Rheum Assn Report, June 6, 1952
157 KUZELL, W C Ann Review Med , 2, 367, 1951.
158 KUZELL, W C , SCHAFFARZICK, R. W Stanford Med Bull , 8, 125, 1950
159 KUZELL, W C , SCHAFFARZICK, R. W , BROWN, B , and MANKLE, E A Jour [illegible]
[illegible] , 1951
Ann
[illegible] Company,
1952
160 SPIES, T D., STONE, R E So. Med Jour , 42, 1, 1949

later there is a production of fibroblasts, capillaries and infiltration with neutrophils and plasma cells. If the disease progresses to an advanced stage, synovial fibrosis occurs.

Depending upon the virulence of the organisms, their numbers and the resistance of the host, various degrees of joint damage may occur. These may range from a mild arthralgia to an acute purulent arthritis; inflammatory reactions in tendons and muscles have not been infrequent.

Postmortem examinations have been made upon joints which were the seat of an acute gonorrheal arthritis at the time of death of the patient [50] In one of these in which gonococci were found in the joint fluid, the synovial membrane was completely destroyed and was replaced by granulation tissue which contained neutrophiles, lymphocytes, macrophages and plasma cells; a perivascular infiltration of lymphocytes was noted in the deeper layers of the synovia. Such inflammatory lesions in the synovial membrane appear to be much less intense in gonorrheal arthritis with sterile synovial fluid

The degree of destruction of articular cartilage varies markedly in gonorrheal arthritis It appears to be greatest in the wrists, phalanges, hips and ankle joints The lessened damage in the knee joint may be due in part to the large amount of fluid containing antitryptic substances which may collect there. The articular cartilage of the knee joint may be pro- [illegible]

such an exudate contains a large number of leukocytes and a relatively small amount of synovial fluid

Ankylosis is not uncommon in improperly treated cases of gonorrheal arthritis This results from periarticular fibrosis or adhesions between the articular cartilages. Adhesions between the patella and the femur result in stiffness of the knee joint True bony ankylosis is uncommon except in the wrists.

Synovial fluids infected with gonococci do not show active bactericidal antibodies but the blood from such persons is frequently bactericidal for the same organism.[31] However, sterile synovial fluid in cases of gonorrheal arthritis is actively bactericidal for the homologous strain of gonococci due to [illegible]

in

Mucin may be a factor in favoring the localization and survival of these organisms in the synovial fluid and tendon sheaths[47] as gonococci seem to localize and survive in areas containing large amounts of mucin (urethra, [illegible]

is very infrequent [46]

A chemically defined medium has been described for the cultivation of the gonococcus [31]

Antibodies appear in the blood during the course of a gonococcic infection The bacteriolytic titer of the blood increases during the course of the disease in patients with gonorrheal arthritis.

77 Lichtwitz, L · Bull New York Acad. Med, 10, 306, 1934.
78 Linton, R R, and Talbott, J H. Ann Surg, 117, 161, 1943
79. Lockie, L M · Ann Int Med, 13, 755, 1939
80 ———: Clinics, 1, 571, 1942.
81. Ludwig, A. O, Bennett, G A., and Bauer, W.· Ann Int Med, 11, 1248, 1938
82 MacLeod, I C, and Phillips, L. Ann Rheumat Diseases, 6, 224, 1947.
83 Mathieu, L, Collesson, L, and Choltus, R.: *Ann de med, 35, 124, 1934*
84 McClure, C W: Med Clin North America, 3, 957, 1920.
85. McCracken, J. P · Bull. New England Med Center, 3, 249, 1941
86. McCracken, J P, Owen, P S, and Pratt, J. H.: Jour. Am. Med Assn, 131, 367, 1946.
87 McEwen, C Jour Mt. Sinai Hosp, 8, 854, 1942.
88 Moore, N. Trans. Path Soc, London, 33, 271, 1882
89 Neel, J. V.· Medicine, 26, 115, 1947.
90 Neuwirth, E Arch Int Med, 72, 377, 1943
91. Nishnevich, M. Y: Klinicheskaya Meditaina, 13, 1819, 1935.
92. Nissé, B. S: Clin Jour, 64, 71, 115, 1935.
93 Oppenheimer, E. H, and Kunkel, H G: Bull. Johns Hopkins Hosp, 73, 40, 1943.
94 Opsahl, R Acta med Scandinav., 102, 611, 1939
95 Peters, J P., and Van Slyke, D D Quantitative Clinical Chemistry, 1, 2d ed, Baltimore, Williams and Wilkins Company, p 959, 1946
96 Pommer, G Mikroskopische Untersuchungen uber die Gelenkgicht, Jena, Gustav F Fisher, 1929
97 Pratt, J. H. In Cecil, R L, Textbook of Medicine, 2d ed, Philadelphia, W B Saunders Company, p 593, 1930.
98 Price, N. L. Lancet, 1, 22, 1939
99 *Pringle, G L. K · Proc Roy Soc Med, 31, 712, 1938*
100 Polley, H F, Tinney, W S, Hall, B E, and Giffin, H Z Proc Staff Meet Mayo Clinic, 20, 49, 1945
101. Questions and Answers Jour Am Med Assn, 110, 1210, 1938
102 Quick, A J Jour Biol. Chem, 101, 475, 1933; and Med Clin North America, 17, 1325, 1934

, 1946

1936
4th ed,

rest, 27,
749, 1948
113 Med Sci 201, 125 1941
114 Therapy, 22, 462, 1941
115 Proc Genet Soc Amer. in 1949.
116 the Root of the Colchicum Latin, T. Becket and P A de Hondt, 1764
117 Stormer, A · Munchen med Wchnschr, 78, 575, 1931
118 Talbott, J H Bull New York Acad Med, 18, 318, 1942
119 ———: Rocky Mountain Med Jour, *38, 186, 1941.*
120 Lancet 19 645 1940
121. Am Med Assn, 110, 1977, 1938
122 S A · Jour Clin Invest, 14, 411, 1935
123. Taylor's "*Principles and Practices* of Medical Jurisprudence," 9th ed, London, J and A Churchill, 1934, p 784

[illegible]

or rectum).
Onset of joint symptoms *usually* within ten to twenty days after primary gonorrheal infection.
[illegible]

Frequent occurrence of *tenosynovitis about the wrists and ankles*
Occasionally associated with conjunctivitis and iridocyclitis
Isolation of gonococci from joint fluid (by special culture methods).
Increased cell count in synovial fluid.
Decreased sugar content of synovial fluid.
Gonococcus complement fixation test of blood positive in over 80 per cent of cases (if properly performed and repeated); may remain negative for the first four weeks of the disease.

Gonorrheal Arthritis Usually Affects Multiple Joints.—*In approximately 80 per cent of the cases, the disease is polyarticular* at its onset. This is not generally appreciated; unfortunately, the thought is prevalent that gonorrheal arthritis is a monarthritis. This is not so It is true that in most cases, after an acute polyarticular onset, one joint may be affected much more severely than others and the disease may seem to persist particularly *in one joint, such as a knee, ankle or wrist.*

Incubation Period.—The disease most commonly begins from ten to twenty days after the onset of an acute gonorrheal urethritis (or at times a gonorrheal cervicitis, conjunctivitis or proctitis), although gonorrheal arthritis has been noted simultaneously with the appearance of an attack of urethritis; in other individuals, Neisserian arthritis has been reported occurring twenty years after the onset of an attack of gonorrhea, being induced in this instance by instrumentation or pelvic surgery.[38] In 80 per cent of several hundred cases of gonorrheal arthritis the joint manifestations appeared within the first five weeks after the primary infection

Age Incidence.—Gonorrheal arthritis may occur at any age but is most common between the ages of twenty and forty. It has been reported in individuals seventy years of age.[49] The simultaneous occurrence of gonorrheal arthritis in a mother and newborn infant has been recorded,[39] but this is rare.[70] Gonorrheal arthritis is uncommon in infants, occurring in less than 2 per cent of children with gonorrheal vulvovaginitis [23,91] In infants who develop a Neisserian arthritis following a gonorrheal ophthalmia, the former may occur between the fifth day and the fifth week of the disease, but most commonly during the second week.

Gonococcic arthritis has been reported in a prematurely born infant; the organisms in this case probably entered *via* the gingivæ.[25]

PATHOLOGY OF GONORRHEAL ARTHRITIS

The pathologic process in gonorrheal arthritis is similar to that in other pyogenic forms of arthritis.[27] One sees thickening, edema and infiltration of the synovia with polymorphonuclear leukocytes in the early stages while

CHAPTER 42

OTHER FORMS OF METABOLIC ARTHRITIS

By Joseph L. Hollander, M.D.

ALKAPTONURIA AND OCHRONOSIS (OCHRONOTIC ARTHRITIS)

Alkaptonuria was first described by Bodeker[1] in 1858 This condition is characterized clinically by the excretion of urine which becomes darker in

aromatic compound resulting from continued local application of phenol. Patients with this metabolic disorder may have no other symptoms, but in severe or long standing cases the presence of alkapton bodies in the blood may produce *ochronosis*.

Alkaptonuria is considered to be a deficiency disease in which an enzyme, which normally breaks down tyrosine and phenylalanine, is apparently

reveals the condition Sometimes, the urine is very dark when voided In a few instances, the disease may not be detected until a routine urinalysis discloses a reducing substance which proves not to be sugar. If a patient

in normal aining oral the urine. ence of the alkaptonuria in these experimental cases[19]

Garrod[9] reviewed the subject of alkaptonuria and noted that it had been known to occur as early as 1584 The reducing substance in the urine in these individuals was described in 1858 by Bodeker,[3] and Brune[5] reported

tinued and severe alkaptonemia and is characterized by pigmentation of certain tissues of the body, more marked in the cartilages of the ears, nose and joints, in the sclerae and cornea of the eye, sometimes the intervertebral discs, and, in rare cases, pigmentation of the skin. Ochronosis may have no

Differential Diagnosis

	Gonorrheal arthritis	Rheumatic fever	Rheumatoid arthritis
History of gonorrhea	Usually within several weeks preceding joint pains	Usually negative	Usually negative, possibly coincidental.
Synovial fluid	Gonococci demonstrable by smear or culture (in at least 30 per cent of cases).	No gonococci	No gonococci
Synovial fluid sugar	Usually low 0-97 mgs. per cent Average 20 mgs per cent	Normal	Normal
Electrocardiogram	Usually no changes	Evidence of cardiac involvement in almost every case if serial examinations are made	Usually negative
Involvement of pleura or lungs	None	Not uncommon.	None
Presence of serous conjunctivitis	In about 20 per cent of cases	Very uncommon	Absent
Preceding upper respiratory infection	In about 10 per cent	In most cases	Frequent
Roentgen-rays	May show rapid bony destruction	Usually negative except for soft tissue swelling	Characteristic may be negative early, later generalized osteoporosis, etc.
Permanent joint damage	May be marked—in one or two joints	Usually none	May occur in many joints
Age at onset	Most often 20 to 40 years, may occur at any age	First attack under 15 years in 90 per cent, may occur at any age	90 per cent of cases after age of 15
Tenosynovitis	Present in almost 50 per cent of cases	Usually absent	Usually absent
Subcutaneous nodules	Absent	Present in about 20 per cent of cases	Present in about 20 per cent of cases, may persist for weeks
Agglutination reaction with hemolytic streptococcic	Usually negative	Usually negative	Strongly positive in most cases
Antistreptolysin titer	Normal	Increased	Normal
Response to penicillin or streptomycin	Excellent	None (May be made worse in acute stage by sulfonamides)	None
Response to salicylates	Not dramatic some relief of pain and some reduction of temperature	Usually dramatic with complete relief of pain and return of temperature to normal if adequate doses are used	Temporary relief, but no dramatic response
Complement fixation test (Gc)	Positive in over 80 per cent of cases if repeated every 10 days for 3 examinations	Almost always negative	Usually negative unless there is previous history of gonorrhea

167. Spitz, H, Steinbrocker, O., Schwartz, S, and Schittone, M.: Am. Jour. Med, 6, 513, 1949.
168 Steinbrocker, O, Berkowitz, S, Carp, S, Ehrlick, M, Elkind, M: Am Rheum. Assn. Report, June 6, 1952
169. Suttenfield, F D · Geriatrics, 6, 96, 1951.
170 Talbott, J. H, Bishop, C, Norcross, B M, and Lockie, L M.· Trans Assn Am Phys, 64, 372, 1951
171. Thorn, G W, Forsham, P. H., Prunty, G, and Hills, A G: Jour. Am Med Assn, 137, 1005, 1948.
172 Thorn, G. W, Forsham, P H, Frawley, S. R, Hill, M, Roche, D, Staehlin, D, and Wilson, D L · New England Jour. Med, 242, 783, 1950.
173 Wolfson, W Q, Hunt, H D, Levine, R., Guterman, H. S, Cohn, C., Rosenberg, E. F, Huddlestun, B., and Katoda, K.: Jour Clin Endo, 9, 749, 1949
174 Wolfson, W Q, Cohn, C Proceedings First Clinical ACTH Conference, Philadelphia, Balkiston Company, 1950
175 Wolfson, W Q, Robinson, W. D Jour Lab Clin Med, 38, 961, 1951
176 Wolfson, W Q, Cohn, C, Levine, R, Rosenberg, E F., Hunt, H. D, and Guterman, H S in "Rheumatic Diseases," Philadelphia, W B Saunders Company, 1952
177. Wolfson, W Q, Guterman, H S, Levine, R, Cohn, C., Hunt, H D, Rosenberg, E F Jour Clin Endo, 9, 497, 1949
178. Wolfson, W Q, Hunt, D, Cohn, C, Robinson, W. D, and Duff, I F: Jour. Mich State Med Soc, 49, 1058, 1950
179 Wrigley, F Ann Rheum Dis, 9, 38, 1950
180 Yü, T F. Am Jour Med, 9, 799, 1950
181. Yü, T. F., Gutman, A B Am Jour Med., 11, 765, 1951

Usual Clinical Picture.—An individual about thirty-five years of age contracts an *acute gonorrheal urethritis*. *Two or three weeks* later, acute *polyarticular arthritis* develops There is a dull aching pain at the onset which is exaggerated by motion. With involvement of the synovial tissues, an *accumulation of synovial fluid* results; as much as 165 cc. of fluid may be aspirated from one of these joints.[64] *Pain* later becomes *severe* and may be excruciating so that the affected part may appear paralyzed because of the desire of the patient to splint it. There is *swelling, redness, tenderness* and *marked limitation of motion of several large joints*, such as the knees, ankles, elbows, etc.

sedimentation rate. As a general rule, the joint disturbance does not tend to become migratory unless the primary focus (urethra, etc.) is subjected to trauma. After the first several days of the disease, the multiple joint

properly treated. The surrounding muscles undergo marked atrophy, because of the immobility of the affected parts due to extreme pain on motion Untreated, the infection leads to destruction of the articular tissue with varying degrees of ankylosis of the joint resulting. The outcome of the disease depends upon the severity of the infection, the resistance of the patient, the promptness of adequate penicillin therapy, reinfection with gonococci and the ability to clear up the original focus of infection Regardless of treatment typical gonorrheal arthritis generally runs its course in three to four months It generally does not remain active indefinitely in the joints initially affected, and there is no tendency for the disease later to involve progressively new joints.

DIFFERENTIAL DIAGNOSIS

Acute gonorrheal arthritis is often confused in its early stages with the acute rheumatoid arthritis [illegible] *genito-urinary tract.* arthritis the focal g [illegible]

states,[44] "Rheumatoid arthritis precipitated by gonorrhea may, like true

tends to be more symmetrical than that of gonorrheal arthritis and, in rheumatoid arthritis, the smaller joints are more commonly affected. Fever may or may not be present for a few days. Synovial fluid cultures are negative for gonococci. There is no unequivocal response of the joints to antigonococcal therapy. There is usually a gradual progression of the disease; new articular sites may become affected even during antigonococcal treatment and even after the genital gonorrhea is cured; in other words, a

GAUCHER'S DISEASE

This rare disease of childhood was first described by Gaucher in 1882 as a primary epithelioma of the spleen Gaucher's disease is a familial disorder of lipoid metabolism, characterized by deposition of kerasin in the reticulum cells of the reticulo-endothelial system. Such cells are called Gaucher cells. Most of the patients are Jewish. The *symptoms* are *most often* encountered *in childhood and adolescence.* The disease may involve any part of the reticulo-endothelial system· spleen, liver, lymph nodes, bone marrow, etc

[illegible]

irregularity of the epiphyseal line.[14] In adults, the hip lesions may present symptoms and signs of degenerative joint disease (osteo-arthritis) and roentgen evidence of Gaucher's disease without symptoms or signs pointing to the hip.

The onset of the disease is often associated with a low grade *fever* and intermittent *pain* in various bones and joints and one finds the *enlarged spleen, liver* and *secondary anemia*, often with leukopenia and thrombocytopenia, brown pigmentation of the skin and hemorrhages.

In 1 case reported by Reed and Sosman[15] the disease began with a migratory polyarthritis, ecchymoses, and spindling of the finger joints Roentgenograms showed narrowing of the joint spaces, and multiple cystic changes in the femoral heads Biopsy of bone marrow from an involved area revealed Gaucher cells

Needle biopsy of *the sternal marrow* will nearly always reveal the diagnosis by the finding of the large typical Gaucher cells Roentgenograms of involved bones show extensive mottling[11]

During the acute symptoms, rest is essential with or without traction or immobilization of the hip in a spica. In childhood, prolonged rest is indicated to minimize deformity Physical and roentgen therapy have been of very little if any benefit [illegible] nent least [illegible] the [illegible] in a woman of thirty-three with this disorder and removed 6 loose sequestra from the partly destroyed acetabular wall, this patient made an uneventful recovery from the operation

The disease may persist for years The only treatment of value is splenectomy, but the operative mortality rate is high. The prognosis in Gaucher's disease is poor Death eventually comes from hepatic failure, when infiltration with Gaucher cells is massive

Examination of the Joint Fluid.—Examination of the joint fluid is of great importance in suspected gonorrheal arthritis. A diagnosis of proven gonorrheal arthritis can only be made by obtaining the gonococcus from the joint fluid or articular tissues. The cell count of the synovial fluid is in-

gonococci is Difco Proteose Peptone No 3 Agar enriched with 2 per cent hemoglobin or with 12 per cent horse blood. Growth is stimulated in an atmosphere enriched with carbon dioxide.[42]

For further identification of the gonococci, fermentation reactions and agglutination with antigonococcus diagnostic serum may be performed.

Gonococcus Complement Fixation Test.—This is of doubtful value This test may remain positive for as long as fifty years after the initial genital infection.[31] False positives are recorded in about 10 per cent and false negatives in another 20 per cent About all that can be said for this test is that, when a negative becomes positive while the patient is under observation, one may assume the presence of a gonorrheal infection. This information is usually more readily obtained by smear and culture.

Skin Tests for Gonorrhea.—Skin tests are of uncertain value, and are not considered valuable diagnostic aids at present.[42]

Roentgen Examinations in Gonorrheal Arthritis.—There are no path-

tion are often seen in roentgenograms in acute gonorrheal arthritis Other

arthritis Several other popular misconceptions may be mentioned. It was taught that involvement of the spine, sternoclavicular joints and temporomandibular joints by gonorrhea was more common than by other forms of arthritis As a matter of fact, gonorrheal spondylitis is uncommon, the sternoclavicular joint is involved in less than 3 per cent of cases of gonorrheal arthritis,[44] and the temporomandibular in only 2 per cent of cases

GONORRHEAL TENOSYNOVITIS

Gonorrheal tenosynovitis is common in association with gonorrheal arthritis In one series, it was

synovitis is more frequent in

other type of arthritis and is a

symptoms of itself, but in time the pigment deposits in the joint cartilage produce extensive degeneration resulting in a special form of arthritis—"Ochronotic or Alkaptonuric Osteoarthritis."

Vir[illegible]

a de[illegible]

acid [illegible]

Ochronosis has been reported in 89 cases to date.[11] Ochronotic arthritis has not been present in all. The condition is more common in men than in women and pigmentation tends to become more marked with advancing years. The pigment is usually deposited intracellularly and in the late cases is so dense that the cartilage involved may appear black. Most of the cases show homogentisic acid in the urine and a few show melanin.[22] Since phenol preparations are no longer in use in treatment of chronic ulcers of the skin, this type of pigment is no longer found.

The symptoms are those of a low grade rheumatoid arthritis or degenerative joint disease. The arthritis is chronic with occasional acute exacerbations and synovial effusion.[1] Homogentisic acid has been found in the synovial fluid.[25] At the time the joints flare up, the amount of alkaptonuria becomes increased. Severe arthritic symptoms may be present even though visible pigmentation is absent in the eyes, ear or nose cartilages or skin. [illegible]

case of ochronosis with prostatic calculi has been reported by Young.[25] The stones were removed and homogentisic acid was found both in urine and in synovial fluid from this patient.

The diagnosis is usually made by: (1) the observation of discoloration of the cartilages of the ear and nose, which may be rendered more obvious by transillumination. (2) Discoloration of the eyes or skin, (3) the demonstration of alkaptonuria, and (4) characteristic changes on the roentgenograms.[11] A rapid method for detection of *alkaptonuria* was recently reported.[8,10] A drop of alkalinized urine placed on ordinary sensitized photographic paper instantly turns the paper black.

The *roentgenograms* are quite unusual, and when the spine is involved in ochronotic arthritis the changes are so characteristic that anyone who has seen them once can make the diagnosis of ochronomic spondylitis on the *x*-ray picture alone.[11 12 15] In the spine, the changes consist of marked thinning and calcification of the intervertebral disc, sometimes accompanied by the development of exostoses or ligamentous calcification. Affected shoulders show marked narrowing of the joint space and large exostoses at the margins.

The treatment of this unusual metabolic disorder has been unsatisfactory. Leslie[14] has discussed the use of liver extract, and vitamins C and E. The use of these substances has been unsatisfactory, although in the experimental cases vitamin C had been beneficial.[19] Swirsky[22] claimed that [illegible] decreased the output [illegible] report of one case[5a] [illegible] ochronotic arthritis

picture characteristic of slowly progressive rheumatoid arthritis may develop." At times the differential diagnosis between rheumatoid arthritis precipitated by gonorrhea and gonorrheal arthritis may be so confusing as to warrant a trial of penicillin therapy. *In gonorrheal arthritis, there is usually a dramatic response within the first two or three days of penicillin. Rheumatoid arthritis is unaffected by penicillin.*

Reiter's syndrome (see next chapter, p. 794) which is characterized by urethritis, conjunctivitis, arthritis, and occasionally diarrhea may simulate gonorrheal arthritis. However, gonococci are never found in the genital secretions or in the synovial fluid, and the disease is unaffected by sulfonamides and penicillin.

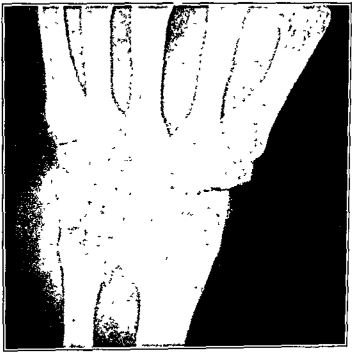


FIG 242 --Gonorrheal arthritis of the wrist in a boy of twenty-one There is very little motion in the wrist The joint spaces between the carpal bones have been entirely destroyed, there is almost complete bony union of all these bones

Acute rheumatic fever, suppurative arthritis caused by staphylococci.

Smears and cultures for gonococci (in joint fluid and in the original focus of infection).

Examination of the Joint Fluid.—Examination of the joint fluid is of great importance in suspected gonorrheal arthritis. A diagnosis of proven gonorrheal arthritis can only be made by obtaining the gonococcus from the joint fluid or articular tissues. The cell count of the synovial fluid is in-

enriched with 2 per cent
rowth is stimulated in an

For further identification of the gonococci, fermentation reactions and agglutination with antigonococcus diagnostic serum may be performed.

Gonococcus Complement Fixation Test.—This is of doubtful value. This test may remain positive for as long as fifty years after the initial genital infection.[31] False positives are recorded in about 10 per cent and false negatives in another 20 per cent About all that can be said for this test is that, when a negative becomes positive while the patient is under observation, one may assume the presence of a gonorrheal infection. This information is usually more readily obtained by smear and culture

Skin Tests for Gonorrhea.—Skin tests are of uncertain value, and are not considered valuable diagnostic aids at present.[42]

Roentgen Examinations in Gonorrheal Arthritis—There are no path-

localized atrophy have been mentioned as suggestive of gonococcal disease[55, 63] Early diffuse atrophy of bone, and marked, rapid bony destruction are often seen in roentgenograms in acute gonorrheal arthritis. Other changes include bony erosions, punched out appearance of the bone, osteolysis and chondrolysis, fibrous or bony ankylosis, and osteophytes or exostoses.

Spur Formation on the Os Calcis May or May Not be Due to Gonorrheal Infection.—Teaching some years ago led one to believe that spur formation on the os calcis (so-called lover's heel) was usually the result of gonorrheal infection This has been shown to be untrue[37] Gonorrheal spurs are uncommon, having been seen in less than 10 per cent of cases of gonorrheal arthritis Several other popular misconceptions may be mentioned. It was taught that involvement of the spine, sternoclavicular joints and temporomandibular joints by gonorrhea was more common than by other forms of arthritis As a matter of fact, gonorrheal spondylitis is uncommon, the sternoclavicular joint is involved in less than 3 per cent of cases of gonorrheal arthritis,[11] and the temporomandibular in only 2 per cent of cases

GONORRHEAL TENOSYNOVITIS

Gonorrheal tenosynovitis is common in association with gonorrheal arthritis.

synovitis is

other type

to infection with dysentery bacilli. They termed this condition the "Conjunctivo-urethro-synovial Syndrome." In the European literature since this time, there has been some tendency to doubt that Reiter's Syndrome is a distinct etiologic entity.[2, 23] Some of the authors considered the syndrome as a complication of infection of the intestinal tract while others felt it was

During the Second World War, many articles reporting additional cases appeared.[2, 5, 17, 19, 20, 25, 27, 35, 39, 42] One of the largest series reported was the one we observed at the Ashburn General Hospital, one of the Army Rheumatism Centers.[19, 20]

ETIOLOGY

The etiology of this disease process must be considered unproved, al-

mentioned spirochaeta forans and various bacterial forms,[28, 32, 46] but none of these have been confirmed as the causative organism. Some[2, 30] have considered the syndrome as allergic in origin. The portal of entry of the infection could be either through the gastrointestinal tract or the genitourinary tract, on the basis of observations of initial symptoms.[19, 26]

Bacillary Dysentery and Uro-Arthritis.—Young and McEwen[52] found 10 cases of urethritis and arthritis among 176 cases of bacillary dysentery in Algeria in 1943. Seven of the 10 also had conjunctivitis. Four additional cases developed arthritis following dysentery, but had no urinary or conjunctival involvement. These authors ascribed the disease to the dysentery infection, even though the stool cultures in all of the 7 cases who exhibited the "classic triad" were negative for the dysentery group, and serum agglutination titers against dysentery organisms were not remarkably high. Short,[43] in considering these cases, believed the Reiter's Syndrome a secondary infection.

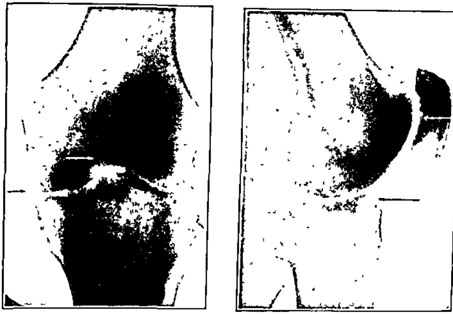
An excellent monograph by Ilmari Paronen entitled "Reiter's Disease—a study of 344 cases observed in Finland" has recently appeared as a supplement to Volume 130 (1948) of Acta Medica Scandinavica. The reader is referred to this one hundred and twelve page monograph for an extensive

tinations for Flexner organisms were high in less than one-half of the sera tested. Agglutinations for Flexner organisms on the aspirated joint fluid

tenosynovitis is present without evidence of arthritis. The most commonly affected tendon sheaths are those of the extensors of the palm and wrist; other
the dorsum
and the radi

In acute gonorrheal tenosynovitis, the pus is greasy, gray and translucent.[30] *Suspect this diagnosis in any acute tenosynovitis with no history of antecedent trauma and especially if there is a focus of gonorrhea.* The diagnosis is made by smear or culture of the pus in the tendon sheath
by

pain occurs on movement This may be acute or chronic. Systemic re-



A B

FIG 243 —Gonorrheal arthritis, lateral view. Areas of erosion indicated by the arrows B, Gonorrheal arthritis, antero-posterior view. (Holmes and Robbins)

action
rare.[5]
type of
results are obtained by splinting and adequate dosage of penicillin.

SUPPURATIVE GONOCOCCIC MYOSITIS

Linner described a case of acute gonorrheal urethritis complicated by suppurative myositis and purulent arthritis.[59] Suppurative gonococcic myositis is an infrequent complication.[45,65,80] In the reported cases, the interval between the genital infection (which usually preceded the abscess) and the suppurative myositis varied from several weeks to several years Primary multiple abscesses were less common than abscesses by direct

CHAPTER 44

REITER'S SYNDROME (INFECTIOUS URO-ARTHRITIS)

By JOSEPH L. HOLLANDER, M.D.

THE symptom complex of non-gonococcic urethritis, conjunctivitis and arthritis (so-called Reiter's Syndrome) has received a great deal of attention during the past ten years, particularly from observers in the Armed Forces.[19,20,26,39,42,52] That this symptom complex is a clinical entity is now fairly generally accepted. The clinical pattern may be quite variable, ranging from a mild "non-specific" urethritis with transitory conjunctival irritation and arthralgia, to a very severe and debilitating disease process with a marked purulent urethritis, prostatitis, cystitis, conjunctivitis, stomatitis, polyarthritis, balanitis, and keratodermia blennorrhagicum. Just where Reiter's Syndrome begins and ends has been rather indefinite, but most observers have regarded occurrence of the so-called "classic triad" of

agents

The acute febrile onset, leukocytosis, purulent exudates, prompt recurrence of a subsiding process upon prostatic massage, and the self-limited complex urinary be considered much more descriptive of the process than the name Reiter's term for the Blennorrheal that filtrable microorganisms of the pleuropneumonia group are closely associated with this form of arthritis, whether or not the conjunctivitis or skin lesions occur.

Historical.—*Reiter*[37] *in 1916 first described a disease characterized by* arthritis, urethritis and conjunctivitis, not caused by gonorrhea, which he had noted in a German officer following a brief attack of abdominal pain and diarrhea. The purulent urethritis with perimeatal inflammation followed the abdominal symptoms by about eight days. The conjunctivæ became red and swollen with purulent discharge, followed by iritis. The *patient was quite ill with continued remittent fever.* Acute pain and swelling was noted in several weight-bearing joints. The course of the disease appeared to be self-limited and was not influenced by either neosalvarsan

extension from an infected joint These abscesses occur most frequently in the thigh, calf, posterior axillary fold, psoas major, latissimus dorsi, posterior tibial and masseter muscles.[82,95,101] Surgical incision and open drainage with daily irrigations have been the most successful treatment to date.[77] In Linner's case, daily doses of sulfathiazole did not prevent the development of suppurative myositis and arthritis. These patients with [illegible] ...y, though surgical

[illegible] did not cure the gonorrheal arthritis in Linner's case although only 10,000 units was given every three hours intravenously for a total of 16 doses. There was considerable improvement in the knee joint with a resulting normal temperature, but there was a recrudescence of the knee infection in a week with gonococci found in the thin yellow pus obtained at aspiration at this time. Following the daily injection (for three days) of 8700 units of penicillin in 15 cc. of sterile physiologic saline solution into the knee joint, the affected knee slowly decreased in size and the patient was apparently cured The *failure to obtain a cure in this case with intravenous penicillin is considered* to have been due to the low dosage of penicillin given. Such serious infections are treated today with much larger doses (100,000 units intramuscularly every three hours)

ASSOCIATED FEATURES IN GONORRHEAL ARTHRITIS

Conjunctivitis is more common in gonorrheal arthritis than in most other forms of joint lesions; (it may occur also in postdysenteric arthritis, Reiter's syndrome and rarely in acute rheumatic fever) Ocular signs may assist in diagnosis, these may be found in from 10 to 20 per cent of cases of gonococcal arthritis The most common is a catarrhal conjunctivitis which may occur simultaneously with or may precede the joint phenomena This conjunctival exudate is sterile. The ocular lesion may disappear before the onset of the arthritis and usually results in no permanent damage In some cases one observes an irido-cyclitis, which is a serious complication; permanent impairment of vision may result. The latter complication is found in approximately 3 per cent of cases of gonorrheal arthritis, while a conjunctivitis may be noted in as many as 15 per cent.

Keratodermia blennorrhagicum, a rare form of skin eruption, has been seen in approximately 3 per cent of patients with gonorrheal arthritis[12] The lesions, which are most common upon the plantar surfaces of the feet, may also occur on the [illegible]
with sharply demarc[illegible]
center of the lesion [illegible]
syndrome and also in pustular psoriasis Although it has been reported[24] that penicillin favorably alters the course of this complication, it has still not been ascertained that this condition is actually a part of true gonococcal arthritis (*see* Chapter 44)

Acute hemorrhagic glomerulonephritis is also a rare complication

Cutaneous Manifestations.—Keil[53] and Lichman[55] have emphasized the frequency and diagnostic significance of cutaneous manifestations which may accompany gonorrheal arthritis. These include petechiae and small

That Reiter's Syndrome can follow a bacillary dysentery is undoubted, but there are many cases in whom no diarrhea occurs, and from whom stool cultures and agglutination reactions are negative for dysentery organisms Only 9 of our 53 cases at Ashburn General Hospital[20] had *any* diarrhea preceding or with the illness, and stool cultures and agglutination reactions for the dyse [illegible] nce, more (14) o [illegible] been apparently [illegible] the syndrome [illegible] dysentery bacilli as causative organisms was the minor epidemic of nonspecific urethritis which was noted at an Army camp in Texas in 1942[20,21] None of these 23 patients had diarrhea nor could gonococci be recovered from the urethral discharge. Five of these patients developed typical Reiter's Syndrome It was reported to us that there were others of the group who had transient arthralgias or mild arthritis. A similar small outbreak on Attu in 1944 was reported to us by one of our patients who had developed his disease at that remote outpost.[21]

The Rôle of Pleuropneumonia-like ("L") organisms in Uro-Arthritis.—Organisms belonging to the pleuropneumonia group ("L" organisms) were first cultured from the human genito-urinary tract by Dienes and Edsall in 1937.[8] These filtrable micro-organisms are known to cause important diseases in animals,[40] and are now suspected of being the causative infec- [illegible]

cause of an important epidemic disease in cattle: bovine pleuropneumonia In 1923, another organism of similar characteristics was discovered as the cause of the disease known as agalactia in goats and sheep, and since that time other members of this group have been discovered in rats, mice, dogs and man[40]

The organism has properties similar to the viruses, being filtrable and invisible within infected tissues and fluids[9] "L" organisms, however, can be grown on lifeless mediums, providing the medium contains serum[9] Colonies of these organisms may be identified on the medium if appropriately stained[4] and viewed under the high power microscope The organisms themselves are quite pleomorphic as studied under the electron-microscope.[10] The colonies grow slowly, and are difficult to identify except by special techniques[8] Organisms of similar properties have been discovered in cultures of various bacteria[7] Some investigators now consider such "L" colonies as variant growth forms of these bacteria[9,11] The pathogenic members of this "L" group are apparently quite specific, and human strains do not appear pathogenic for animals Of major interest in all "L" organism infections is the usual predilection for synovial tissue and mucous membranes[40] The infection may be either an acute suppurative process, or, as in the epidemic polyarthritis of mice, often a subacute proliferative [illegible]

human sources, such as conjunctivæ, stools, skin lesions, etc., have been

red sore spots (at times with a central white necrotic region) and hemorrhagic purulent vesicles or bullæ, surrounded by an erythematous areola.

Frequency of Bacteremia in Gonorrheal Arthritis.—Gonococci have been recovered from the blood stream in 4 per cent of these patients; the mortality in the group with bacteremia approximated 60 per cent in the era before the use of sulfonamides and penicillin. In about 40 per cent of the patients with bacteremia, endocarditis is present.

One of the striking features of patients with such bacteremia is the maculopapular eruption, which may become hemorrhagic, vesicular and then pustular This appears on the trunk but is more intense on the extremities Enlargement of the spleen and liver, jaundice and thrombocytopenia have been seen occasionally in these individuals.

Gonococcus Carriers.—Carpenter and Westphal studied *the problem of the gonococcus carrier* and showed conclusively that a person may have a low-grade carrier state for years which is difficult to overcome, such a focus for the possible development of gonorrheal arthritis can smolder indefinitely.

[illegible]

the latter may cause an increase in the severity of a latent gonorrheal infection. One patient whose history included a gonorrheal urethritis at the age of twenty, *developed an acute exacerbation of his gonorrheal* [illegible]

[illegible]

patients, 10 were women who knew of no gonococcal infection before their admission to the hospital.

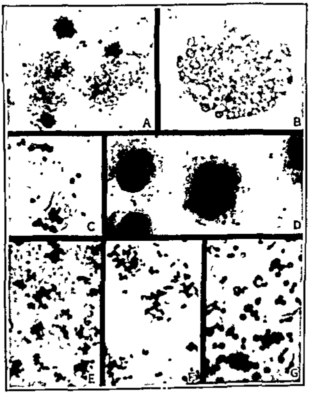
TREATMENT OF GONORRHEAL ARTHRITIS

Penicillin is the drug of choice in all gonorrheal infections, including arthritis By the use of penicillin most cases of gonorrheal arthritis may be controlled very promptly.[42,44] However, one must remember that auxiliary treatment is of importance, this includes rest of the affected parts, splinting, sedation, physical therapy, etc.

Treatment of Gonorrheal Arthritis
Penicillin—in adequate dosage
Rest of the affected joints by splints and prevention of deformity during the acute phase of the disease.
Analgesics (aspirin, codeine and morphine if necessary)
Aspiration of affected joints and introduction of penicillin into the joint cavity
General care of the patient, careful follow-up physical therapy

Penicillin, Streptomycin, Sulfonamides.—Gonococci are among the more penicillin sensitive bacteria and since 1943 penicillin has entirely

were significantly elevated in less than one-third. In no case was the dysentery organism grown from the exudates during the Reiter's Syndrome and in only 7 of 290 stool cultures was the Flexner bacillus grown. No cultures for pleuropneumonia-like ("L") organisms were made. In spite of these equivocal results the author concludes that "Reiter's Syndrome thus seems to occur only after dysentery and the dysentery bacillus seems to be the causative factor also in such cases in which no dysenteric infection can be demonstrated by clinical means."



colonies consisting of small *bacillary* *fo*
(× 125) *E* shows a wet, stained-agar preparation of young colonies of pleuropneumonia- ... visible because
reparation of
which the

replaced the sulfonamides which have become increasingly less effective

practically always respond to a second In gonorrheal arthritis the penicillin dosage must be much higher than in simple gonorrhea, and though the minimum dose may vary from patient to patient 100,000 units every three hours is usually satisfactory. An unequivocal decrease of pain and swelling in the affected joint is almost invariably noted with the first two to three days, but to insure complete eradication of the articular infection treatment should be continued for a minimum of from seven to ten days. Intraarticular penicillin is rarely necessary for penicillin has been shown to penetrate readily into synovial fluid following its intramuscular administration[2] *If intraarticular penicillin is resorted to, it should not be in place*

sensitive to penicillin in dilutions of 1/10,000.[12, 31] Bahn *et al.* were able in vitro to make 5 strains of gonococci resistant to penicillin[1] However, it was much more difficult than producing sulfathiozole resistant strains and thus they believe less likely to develop spontaneously and become established in the population at large.

Aureomycin, terramycin, chloromycetin, and streptomycin are also effective against gonococci Streptomycin has been shown effective not only against naturally occurring gonococci but also against those made penicillin-resistant in vitro[10, 61]

The sulfonamides have become increasingly less effective in gonorrhea,

some cases this may be accomplished by cotton padding and wood or pillow splints When possible, one may use Castex or plaster splints. These are well-padded with cotton so that no pressure points exist. The splints are held in position by adhesive strapping, light gauze bandage or tapes.

The patient notices relief of local pain as soon as splints are applied because of the absence of motion of these joints Such splints should be

dividuals feel that they can be broken up if the splints are left in place more

logical ch[illegible]
process.[5]
ported,[6]
other infections, as so far studied. Cells from the genito-urinary tract have been negative for inclusion bodies,[1] as have scrapings of the skin lesions and conjunctival epithelium.[1] No work has as yet been reported on the pathological changes occurring in the viscera, particularly the intestine, but descriptions of the pathology found in the joint tissues[1,19] and in certain of the skin lesions[25] have been made available through several reports.

Joint Pathology.—During an acute attack of uro-arthritis (Reiter's Syndrome) arthrotomy of the knee was performed in the *first week* of the disease.[21] Although this patient, one of the 53 observed at Ashburn

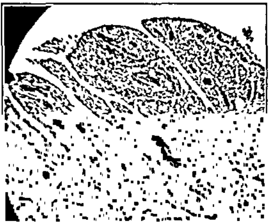


FIG. 245.—Section of synovium from Reiter's Syndrome (seven months). Tissue from knee shows congestion of vessels, and cellular infiltrations.

General Hospital in 1945, was experiencing severe pain in the knee with considerable edema, increased heat, and tenderness of the joint, the only change ob[illegible]

this early case.

Bauer and Engelman, in 1912,[1] described a synovial biopsy from the knee of a patient in the *thirteenth week* of his arthritis from Reiter's Syndrome. Gross examination of the synovium at that time showed marked injection but only slight thickening. A few areas of villus formation were noted in the supra-patellar pouch and a portion of this, studied microscopically, showed that the intima of the synovium was several layers deep and contained a moderate excess of lymphocytes, plasma cells and a few leukocytes. No exudate on the surface was seen. A scant diffuse infiltration of inflammatory cells was present in the subintimal layer of the synovium. Hyperemia and edema were noted. A few perivascular focal collections of round cells were seen in this region. The hyperemia was noted as the most striking feature.

mg. 1/32 1/20 gr.). The latter does not produce as much gastric distress as occasionally occurs with morphine; in addition, it is not so habit forming as morphine. If the pain is so severe that the patient requires morphine for relief (although the part has been placed at physiologic rest) this drug should be given

Aspiration of the Affected Joints.—Aspiration of the joints may be done to relieve pain, to protect the intra-articular tissues from contents which would destroy cartilage, for diagnostic purposes, to reduce the formation of adhesions, to prevent undue stretching of the joint capsule, and prior to the introducti[illegible]

Criteria of C[illegible]
ported in cas[illegible]
covery has also been observed in patients with blood stream invasion by the gonococcus with symptomatic treatment only.[3] The following points must be observed before a cure of genito-urinary gonorrhea can be accepted: cessation of the disease clinically; clear urine without shreds, or absence of gonococci in these shreds if present, several negative smears and cultures for gonococci; a normal prostate whose secretion contains no excess of leukocytes; absence of adnexal tenderness, and no cervical irritation in women; no urethral flare-up following alcohol, sexual excitement, instrumentation, or menses[39]

General Care of the Patient.—[illegible]

required. As soon as the temperature has returned to normal, nourishment should be forced.

If the individual is anemic, iron is given by mouth (such as ferrous sulfate, 0 2 gm (3 gr.), 4 times per day), transfusions may improve the general condition but are seldom needed. In adults, 400 or 500 cc. of blood may be given every several days if required

After the acute stage of the disease has passed, physical therapy (including heat, massage, baking, baths, etc.) will be required (see chapter on Physical Therapy, p 405). The responsibility of the physician does not cease at the end of the period of penicillin administration. Proper support for the affected joints, rest, heat, exercise and massage must be prescribed as required The progress of the infection may be followed by means of the sedimentation rate

If weight-bearing joints are affected, walking should not be permitted until all evidence of inflammation in the joints has subsided.

[illegible]

this heroic treatment, the above list of references is given.

Penicillin Resistant Gonococci.—The gonococcus is one of the most highly pencillin-sensitive bacteria and to date no penicillin-resistant gonococci have been encountered at the Massachusetts General Hospital, the Boston City Hospital, or the Boston Dispensary. Kane, in a review of [illegible] that failed to respond to [illegible] s leading to an erroneous [illegible] 1) wrong diagnosis Not [illegible] or Reiter's Disease; (2)

negative on culture except for the *synovial fluid* from *two patients* with *Reiter's Syndrome.*[10] Dienes and his co-workers were able to culture "L" organisms from the genito-urinary tract in 58 out of 244 women. In many of these, the organisms were present in conjunction with other pathogenic bacteria but in some with "non-specific leukorrhea" the "L" organisms were found in pure culture

In male patients having evidence of genito-urinary tract infection, the organism was recovered from the urethral or prostatic secretion in 115 cases[12] Of the patients from whom positive cultures were obtained, 27 had associated arthritis. The clinical picture was characteristic of Reiter's Syndrome in 9 of these, consisting of urethritis, conjunctivitis, and arthritis. Nine more cases had sub-acute or chronic joint involvement, but a total of 18 had acute arthritis resembling infectious arthritis from other organisms. *One-half* of the cases of *acute* infectious arthritis apparently from this cause, therefore, were typical Reiter's Syndrome, whereas the other half had *no conjunctival involvement.* In our series of 53 cases at the Army Rheumatic Disease Center,[20] 32 of the patients with infectious uro-arthritis ... wenty-one did not, but

... f "L" organisms in the genito-urinary tract and the development of disease has not been so apparent.[10] In the 58 women from whom positive cultures were obtained by Dienes, only 9 had joint complaints, and 6 of these had a chronic arthritis One of the other 3 women developed an acute arthritis two weeks after *marriage, and it is quite noteworthy that her husband also developed* joint symptoms six weeks later. "L" organisms also were cultured from his prostatic secretion. Dienes and his co-workers have seen 2 other female patie ... ve for "L" ... Lind[4] cultu ... n men had ... it the disease may be contracted venereally is suggested by the report of Dienes *et al.*[10] that at least 7 of the men from whom positive cultures were obtained had developed the urethritis within a short time after sexual exposure. Another case is reported by Rosenblum[29] of Reiter's Syndrome occurring after sexual exposure. This acute attack was followed by complete remission with recurrence after two-and-one-half years, again following sexual exposure with the same woman. About half of our 53 patients[20] however, denied sexual exposure within one month of onset. In a few of these, the gastrointestinal tract may have been the portal of entry of the infection because the process began with diarrhea, but the mode of transmission in most cases is unknown. It is conceivable that an infection with pleuro-

... *invasion, variation of* pathogenic bacteria into an "L" form,[13] or a sensitization reaction to an infection is still unknown.

PATHOLOGY

Although many studies on exudates have been reported, little is known of the actual pathology in the tissues in Reiter's Syndrome The patho-

inadequate dosage of penicillin, (3) gonorrheal abscesses of the genitourinary tract (periurethral, prostatic, vaginal, tubes, ovaries) to which penicillin may not penetrate and may require drainage before cure is obtained; (4) reinfection. In spite of occasional reports to the contrary it now seems unlikely that naturally occuring penicillin-resistant gonococci will develop However, if gonococci proved by culture to be penicillin-resistant should be found it is probable that one of the other so-called "broad spectrum antibiotics" would be effective Streptomycin, aureomycin, chloromycetin, and terramycin are all effective against gonococci though penicillin is the most effective at this time

BIBLIOGRAPHY

1 Bahn, J M, Ackerman, H, Carpenter, C M Soc Exp Biol and Med, **58** 21, 1945
2 Balboni, V G, Shapiro, I M, Kydd, D M Am Jour. Med Sci, **210**, 588, Nov 1945
3 Bakst, H J, Foley, J A, and Lamb, M E Ann Int Med, **9**, 790, 1935
4 Bierman, W, and Levenson, C L Med Clin North America, **21**, 1709, 1937
5 Birnbaum, W, and Callander, C L Jour Am Med Assn, **105**, 1025, 1935
6 Boak, R. A, Carpenter, C M, and Warren, S L Quoted in Minnesota Med, **19**, 151, 1936
7 Brunet, W M, Salberg, J B, and Koch, R A New England Jour Med, **223** 277, 1940
8 Carpenter, C M, Boak, R A, Mucci, L A, and Warren, S L Jour Lab and Clin Med, **18**, 981, 1933
9 Carpenter, C M, and Westphal, R S Am Jour Pub Health, **30**, 537, 1940
10 Chinn, B D, *et al* Am Jour Syph, **31**, 268, 1947
11 Chwalla, R Wien klin Schnschr, **46**, 169, 206, 1933
12 Combes, F C, Dietrich, C, and Cohen, J Jour Am Med Assn., **114**, 2078, 1940
13 Cohn, A and Seijo, I H Jour Am Med Assn, **124**, 1125, 1944
14 Conrad, C K New York State Jour Med, **36**, 1767, 1936
15 Corbus, B C Illinois Med Jour, **67**, 521, 1935
16 Culp, O S Jour Urol, **43**, 737, 1940
17 Dawson, M H Nelson's Loose Leaf Med, New York, Thomas Nelson & Sons, **5**, 605, 1935
18 Desjardins, A U Med Clin North America, **19**, 585, 1935
19 Desjardins, A U, Stuhler, L G, and Popp, W C Jour Am Med Assn, **106**, 690, 1936
20 Editorial Jour Am Med Assn, **109**, 1044, 1937
21 Fetter, F Weekly Roster and Med Digest, Philadelphia, **33**, 1583, 1938
22 Flippin, H F, Rose, S B, Schwartz, L, and Domm, A H Am Jour Med Sci, **201**, 585, 1941
23 Fraser, C K, and Dye, W J P Jour Am Med Assn, **105**, 269, 1935
24 Freireich, A W, Schwartz, S, and Steinbocker, O Arch Int Med, **79**, 239, 1947
25 Gaing, E, Cossoy, S, and Aguilar, G D An Soc puericult Buenos Aires, **7** 83, 1941
26 Garland, L H Radiology, **25**, 416, 1935
27 Ghormley, R K, and Deacon, A E Am. Jour Roentgenol, **35**, 740, 1936
28 [illegible] Child, **13**, 438, 1947
29
30 [illegible] **221**, 228, 1939
31
32
33
34
35

nuclear leukocytes and lymphocytes. The epithelium around the vesicles showed edema, moderate acanthosis and infiltration with polymorphonuclear leukocytes The horny layer was thickened, parakeratotic and infiltrated with polymorphonuclear leukocytes and lymphocytes The histologic picture was regarded as consistent with keratosis blennorrhagica in its early "pustular" stage.

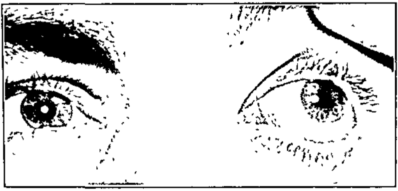


FIG 247 —Early congestion of conjunctival vessels of left eye in a patient with Reiter's Syndrome

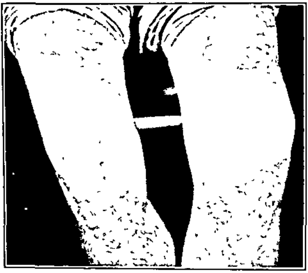


FIG 248 —Inflammation of left knee with effusion in a patient with Reiter's Syndrome

Biopsy of a well-developed pustule from another patient[25] showed the

mg. 1/32 1/20 gr.). The latter does not produce as much gastric distress as occasionally occurs with morphine; in addition, it is not so habit forming as morphine. If the pain is so severe that the patient requires morphine for relief (although the part has been placed at physiologic rest) this drug should be given.

Aspiration of the Affected Joints.—Aspiration of the joints may be done to relieve pain, to protect the intra-articular tissues from contents which would destroy cartilage, for diagnostic purposes, to reduce the formation of adhesions, to prevent undue stretching of the joint capsule, and prior to the introduction of penicillin directly into the joint space.

Criteria of Cure of Gonorrhea.—Spontaneous remissions have been reported in cases of gonorrheal arthritis without specific treatment. Recovery has also been observed in patients with blood stream invasion by the gonococcus with symptomatic treatment only.[3] The following points must be observed before a cure of genito-urinary gonorrhea can be accepted: cessation of the disease clinically; clear urine without shreds, or absence of gonococci in these shreds if present; several negative smears and cultures for gonococci; a normal prostate whose secretion contains no excess of leukocytes; absence of adnexal tenderness, and no cervical irritation in women; no urethral flare-up following alcohol, sexual excitement, instrumentation, or menses.[39]

General Care of the Patient.—The patient should be kept at rest in bed until several days after the temperature has subsided and the joint phenomena have greatly improved. During the febrile stage, a liquid diet may be required. As soon as the temperature has returned to normal, nourishment should be forced.

If the individual is anemic, iron is given by mouth (such as ferrous sulfate, 0.2 gm. (3 gr.), 4 times per day); transfusions may improve the general [illegible] In adults 400 or 500 cc. of blood may be

[illegible] ierapy (including heat, massage, baking, baths, etc.) will be required (see chapter on Physical Therapy, p. 405). The responsibility of the physician does not cease at the end of the period of penicillin administration. Proper support for the affected joints, rest, heat, exercise and massage must be prescribed as required. The progress of the infection may be followed by means of the sedimentation rate.

If weight-bearing joints are affected, walking should not be permitted until all evidence of inflammation in the joints has subsided.

Fever Therapy in Gonorrheal Arthritis.[8, 18, 19, 20, 21, 37, 40, 41, 43, 54, 56, 66, 67, 83, 85, 89, 96]—Modern antibiotics have entirely superseded this form of therapy. Because of its former widespread use, and possible interest in the development of this heroic treatment, the above list of references is given.

Penicillin Resistant Gonococci.—The gonococcus is one of the most highly pencillin-sensitive bacteria and to date no penicillin-resistant gonococci have been encountered at the Massachusetts General Hospital, the Boston City Hospital, or the Boston Dispensary. Kane, in a review of [illegible] one that failed to respond to [illegible] tors leading to an erroneous [illegible]: (1) wrong diagnosis. Not gonorrhea but actually non-specific [illegible]is, or Reiter's Disease; (2)

Biopsy of the knee joint was performed on a patient at the Ashburn General Hospital in the eighth month of the arthritis in a patient having the clinical findings of Reiter's Syndrome.[19] The synovium was congested and reddish-purple in color. There were several small focal collections of fibrinous exudate on the surface of the synovium. Examination of sections of the synovium of the knee showed an intense inflammatory reaction in the superficial synovial layers which did not seem to effect the underlying connective tissue. The synovium was thrown into large club-like projections containing abundant dilated capillaries (Fig. 245). The projections were distended by a heavy infiltration of lymphocytes, with a few plasma cells and neutrophiles The intima was 6 to 10 cells deep. A few perivascular focal collections of lymphocytes and plasma cells could be found. (Fig. 246)

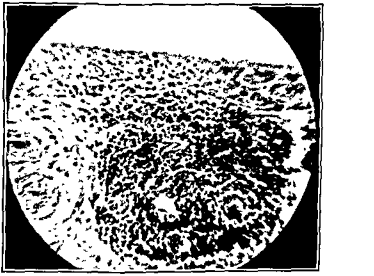


FIG 246 —Perivascular collection of round cells just below surface of synovium of knee

Although we had previously commented[17] that the marked synovial hyperemia, and limitation of the inflammatory reaction to the superficial layers of the synovium appeared distinctly different from the more widespread synovial reaction in rheumatoid arthritis, the type of cellular reaction seen was not unlike the pathologic picture of rheumatoid arthritis This was pointed out to us by Hench,[18] who felt that pathologically no distinct differentiation was possible. Nichols and Richardson,[29] in their classic description of the pathology of arthritis, commented that the modes of response of the synovium to irritation, from *whatever cause*, was limited to the proliferative or degenerative types The synovial response in in- [illegible] this is

[illegible] dermia blennorrhagica with Reiter's Syndrome were taken by Lever and Crawford.[2] A specimen from an early cutaneous lesion showed several small intra-epithelial vesicles filled with degenerated epithelial cells, polymorpho-

89 ——— Ann Int Med, 10, 1014, 1937
90 ———: Am Jour Med Sci, 192, 497, 1936.
91 Su, T. F., and Hu, H T Chinese Med Jour, 50, 583, 1936
92 Thompson, L Am Jour Clin Path, 5, 313, 1935
93 Turner, T B, and Steinberg, T H Jour Am Med Assoc, 124, 133, 1944.
94 Ware, M W Am Jour Med Sci, 122, 40, 1901
95 Warren, C F, Hinton, W A, and Bauer, W Jour Am Med Assn, 108, 1241, 1937
96 Warren, S L, Scott, W W, and Carpenter, C M Jour Am Med Assn 109, 1430, 1937
97 Welton, J P, Stokinger, H F, and Carpenter, C M 99, 373, 1944
98 Wishengard, M Urol and Cutan Rev, 41, 256, 578, 1937
99 Wolbarst, A L Internat Jour Med and Surg, 47, 506, 1934
100 Wynn, W H Lancet, 1, 352, 1905

nuclear leukocytes and lymphocytes The epithelium around the vesicles showed edema, moderate acanthosis and infiltration with polymorphonuclear leukocytes. The horny layer was thickened, parakeratotic and infiltrated with polymorphonuclear leukocytes and lymphocytes. The histologic picture was regarded as consistent with keratosis blennorrhagica in its early "pustular" stage.

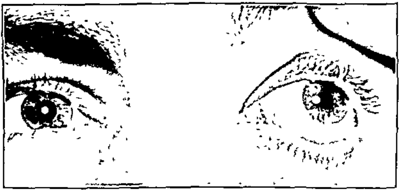


FIG 247 —Early congestion of conjunctival vessels of left eye in a patient with Reiter's Syndrome

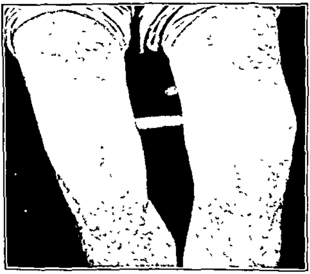


FIG 248 —Inflammation of left knee with effusion in a patient with Reiter's Syndrome

Biopsy of a well-developed pustule from another patient[25] showed the vesicle to contain degenerated epithelial cells, polymorphonuclear leukocytes, lymphocytes and fibrin The surrounding epithelium and underlying corium showed dense infiltration with polymorphonuclear leukocytes and

36. Hench, P. S., and Boland, E. W.: Ann. Int. Med., 24, 808, 1946.
37. Hench, P. S., *et al.*: Ann. Int. Med., 10, 754, 1936.
38. ———: *Ibid.*, 8, 1315, 1935.
39. ———: *Ibid.*, 11, 1089, 1938.
40. ———: *Ibid.*, 12, 1005, 1939.
41. ———: *Ibid.*, 14, 1383, 1941.
42. ———: *Ibid.*, 28, 74, 1948.
43. Hench, P. S., Slocum, C. H., and Popp, W. C.: Jour. Am. Med. Assn., 104, 1779, 1935.
44. Hench, P. S.: Personal Communication.
45. Jeck, H. S.: Nelson's Loose Leaf Medicine, New York, Thomas Nelson & Sons, 1941, p. 2071.
46. Kahlmeter, G.: Reports on Chronic Rheumatic Diseases No. 3, ed. by C. W. Buckley, New York, The Macmillan Company, 1937, p. 21.
47. Kane, L.: Personal Communication.
48. Kapo, P. J.: Am. Jour. Roent. and Rad. Ther., 33, 359, 1935.
49. [illegible]
50. [illegible]
51. [illegible]
52. [illegible] Jour. Clin. Invest., 17, 23, 1938.
53. Keil, H.: Quart. Jour. Med., n.s., 7, 1, 1938.
54. Kendall, H. W., Rose, D. L., and Simpson, W. M.: Jour. Am. Med. Assn., 116, 357, 1941.
55. Key, J. A.: Am. Jour. Roent. and Rad. Ther., 30, 34, 1933.
56. Krusen, F. H.: Proc. Mayo Clin., 13, 297, 1938.
57. Lees, D.: Brit. Jour. Vener. Dis., 8, 79, 182, 1932.
58. Lichtman, S. S.: Jour. Mt. Sinai Hosp., 4, 72, 1938.
59. Linner, J. H.: Jour. Am. Med. Assn., 123, 757, 1943.
60. McEwen, C., Bunim, J. J., and Alexander, R. C.: Quoted by Hench, P. S., Mayo Clinic, Proc., 10, 615, 1935.
61. McLeod, J. W., and others: Jour. Path. and Bacteriol., 39, 221, 1934.
62. Meads, M., and Finland, M.: Am. Jour. Syph. Gon. & Ven. Dis., 30, 586, 1946.
63. Miller, C. P., and Bohnhoff, M.: Jour. Am. Med. Assn., 130, 485, 1946.
64. Myers, W. K., and Gwynn, H. B.: Med. Ann. Dist. Col., 4, 194, 1935.
65. Newburger, B.: Ann. Surg., 84, 879, 1926.
66. Newman, M. K., and Berris, J. M.: Physiother. Rev., 17, 139, 1937.
67. Neymann, C. A.: Artificial Fever, Springfield, Charles C Thomas, 1938.
68. Oard, H. C., Jordan, E. V., and Nimaroff, M.: Jour. Am. Med. Assn., 125, 323, 1844.
69. Pancoast, H. K., and Pendergrass, E. P.: Quoted by Kapo, No. 43.
70. Parrish, P. P., Console, W. A., and Battaglia, J.: Jour. Am. Med. Assn., 114, 241, 1940.
71. Pelouze, P. S.: Journal-Lancet, 56, 307, 1936.
72. ———: Gonorrhea in the Male and Female, 3d ed., Philadelphia, W. B. Saunders Company, 1939.
73. Pepper, O. H. P.: Ann. Int. Med., 3, 328, 1929.
74. Pettit, A. V.: West. Jour. Surg., 45, 637, 1937.
[illegible], 214, 1937.
[illegible] Jour. Dis
[illegible] 1005, 1938
[illegible]
85. Simmons, E. E.: Am. Jour. Med. Sci., 194, 170, 1937.
86. Simpson, W. M., and Kendell, H. W.: Colorado Med., 34, 782, 1937.
87. Spink, W. W., and Keefer, C. S.: Jour. Clin. Invest., 27, 17, 1938.
88. ———: Jour. Am. Med. Assn., 109, 325, 1937.

lymphocytes. Another section from an atrophic area previouly involved, showed thinning of the epithelium and dense fibrosis of the corium.

CLINICAL PATTERN

In most cases the initial symptom of Reiter's Syndrome is urethritis. In about 10 per cent of cases, a mild to moderately severe enteritis may be the first sign of the disease, and in rare cases the first symptom is the conjunctivitis or arthritis [19]

The genito-urinary infection may consist of a slight muco-purulent discharge noted in the morning, or may be an acute, copious purulent discharge with dysuria, hematuria, and frequency of urination The urethritis



Fig. 249 —Swelling of ankles and keratotic lesions of toes in a patient with Reiter's Syndrome

frequently is complicated by prostatitis or cystitis. We have never seen epididymitis Although repeated careful studies of the discharge are made prior to institution of chemotherapy, no gonococci can be seen in the abun- [illegible] The duration of the discharge is variable from a few

tion, the discharge sometimes recurs. While the genito-urinary infection is often troublesome, it is the joint involvement that usually brings the patient to the hospital for treatment.

Like the genito-urinary symptoms, the conjunctivitis is variable in severity and duration Although 32 of our 53 cases experienced this inflammation, in the remaining 21 conjunctival irritation was either so trivial as to pass unnoticed, or was entirely absent, even though the disease was

quite typical in every other respect.[20] The conjunctival inflammation, whenever present, resembles that of acute catarrhal conjunctivitis, or

cases,[19 24] but severe or permanent damage to the eye has not been reported.

One of the most characteristic features of infectious uro-arthritis is the acute, even explosive, onset of polyarthritis accompanied by moderate fever.[19] Weight-bearing joints are most often affected, mostly ankles and knees (Fig 248) Less than half of the patients develop involvement of

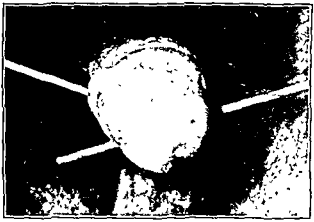


FIG 250 —Circinate lesions on glans penis (balanitis circinata) in a patient with Reiter's Syndrome Note also ulceration around meatus

symmetrical joints,[19] but for example may have inflammation in one ankle, one knee, and perhaps a wrist. Fingers, toes, hips, and spinal joints are rarely affected [19] The frank chill which so often initiates other acute infectious arthritides is seldom, if ever, encountered The affected joints become hot, swollen, and tender. Joint involvement may spread rapidly to new areas, but the transient migratory joint swelling of rheumatic fever is not often encountered, nor is the marked periarticular edema and para-arthritis so often found in gonococcal arthritis usually present The fever in this acute stage is generally recurrent daily to about 101° F and may persist several weeks Anorexia, malaise, tachycardia, and rapid muscular wasting are often seen in the more severe instances. Generalized lymphadenopathy,[28 19] and even splenomegaly [1 22] have been reported, but have not been noted in most cases

Skin Lesions —Skin lesions sometimes complicate the clinical picture of infectious uro-arthritis (Reiter's Syndrome). In 26 of our 53 cases[20] and in many cases reported by others,[20 28] superficial circinate lesions were ob-

within two weeks without recurrence, and the third case seemed to subside

bation

tudies

criti-

cally, but streptomycin therapy is certainly worthy of trial in infectious uro-arthritis[24, 19] Brown *et al*[6] have found that most human strains of

in each was prompt and dramatic, but the symptoms returned in all three after the hormone therapy was stopped Following the initial post-treatment relapse, two of the cases showed an apparently spontaneous subsidence of symptoms, but the third relapsed every time ACTH or cortisone was withdrawn, and appeared chronic From these findings it appears that the hormones cause a prompt suppression of symptoms while being administered, but have little or no effect on the cause or natural course of this usually self-limited disease Certainly the use of ACTH, cortisone, or hydrocortisone should not be recommended for treatment of Reiter's Syndrome except when antibiotics have failed

CONCLUSIONS

There has been much controversy and disagreement concerning Reiter's Syndrome This has stemmed largely from early conflicting reports[12, 31] of cases inadequately studied, and from insistence on the part of some that Reiter's Syndrome must consist of urethritis, conjunctivitis, and arthritis, of others that the syndrome is variable, and of still others that any non-gonococcal urethritis with arthritis represents the Syndrome

This self-limited form of arthritis, with a rather spectacular clinical pattern, has been confused with many other ill-defined diseases, but the development of methods for isolating the etiologic agent, and greater usage of these methods, should help clarify the present confusion If strepto-

careful study is needed

BIBLIOGRAPHY

1 Bauer, W, and Engelman, E P Trans Assoc Am Physicians, 57, 37, 1942
2 Beiglbock, W Deutsche med Wchnschr, 69, 803, 1943
3 Beveridge, W I B Med Jour Australia, 2, 479, 1943
4 Beveridge, W I B, Campbell, A D, and Lind, P E Med Jour Australia, 1, 179, 1946

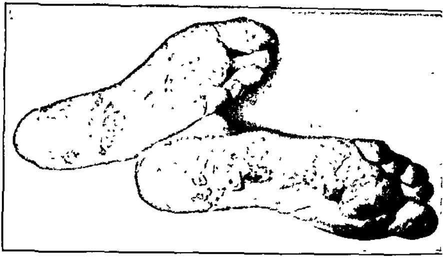


FIG 251 —Keratodermia blennorrhagicum of feet in a patient with Reiter's Syndrome



FIG. 252 —Keratotic and pustular lesions of fingernails in a patient with Reiter's Syndrome

served on the glans penis. (Fig. 250) These lesions were sometimes peri-meatal ulcerations beginning as small blebs, but often took the form of a more widespread balanitis. This "balanitis circinata" has been reported in many of the European and American cases, and in our experience is almost as common an occurrence as the conjunctivitis. The lesions may

5 BROWN, T McP., WICHELHAUSEN, R. H., MERCHANT, W. R , and ROBINSON, L B Am. Jour Med Sci , 221, 618, 1951.
6 COLBY, F H. Jour Urol , 52, 415, 1944.
7 DIENES, L Jour Bact , 50, 441, 1945
8 DIENES, L , and EDSALL, J Proc Soc Exper Biol. & Med , 36, 740, 1937.
9. ———. *Ibid* , 44, 468, 1940
10 DIENES, L , ROPES, M W , SMITH, W E , MADOFF, S , and BAUER, W.: New Eng Jour Med , 238, 509, 563, 1948
11. DIENES, L., and SMITH, W E Proc. Soc Exper Biol & Med , 50, 99, 1942
12 DIENES, L. BERG, R. L , and WEINBERGER, H. J : Ann. Rheum. Dis. 7, 259, 1948.
13 DIENES, L , and WEINBERGER, H J . in "Rheumatic Diseases," W. B Saunders Co , Philadelphia, 401, 1952.
14 FIESSINGER, N., and LEROY, E Bull et mém Soc méd d. hop. de Paris, 40, 2030, 1916
15 FRÜHWALD, R Urol and Cutan Rev , 32, 7, 1928
16 GROSSMAN, C. M , SAPPINGTON, T. S , BURROWS, B A , LAVIETES, P H and PETERS, J. P · Jour. Clin Invest , 24, 523, 1945
17. HARKNESS, A H Brit Jour Ven Dis , 21, 93, 1946.
18 HENCH, P S Personal Communication
19. HOLLANDER, J L , FOGARTY, C W , JR , ABRAMS, N R , and KYDD, D. M : Jour Am Med Assn , 129, 593, 1945.
20. HOLLANDER, J. L . Med Clin. North America, 30, 716, 1946
21 ———: Unpublished Data.
22 JACKSON, W P N Brit. Med Jour., 1, 197, 1946.
23 [illegible]
24. [illegible] 1950
25 [illegible] 1944
26 [illegible]
27 [illegible]
28 [illegible]
29 [illegible] 1909
30 [illegible]
31. [illegible] 950
32. [illegible]
33 [illegible]
34. [illegible] Biol and Med , 62, 8, 1946
35 POWELL, H M , and RICE, R M Jour Lab and Clin. Med , 29, 372, 1944
36 PRESTON, W. S Jour Infect Dis , 70, 71, 1942
37. REITER, H Deutsche med Wchnschr , 42, 1535, 1916.
[illegible] 945
[illegible] 40
[illegible] 45
[illegible] 24, 1043, 1946
[illegible] . Infect. Dis , 79, 134, 1946
[illegible] & Med , 51, 24, 1942
49 WARTHIN, T A Am Jour Med , 4, 827, 1948
50 [illegible] p 73, "Rheumatic Diseases,"
51 [illegible] ERSON-BEGG, A : Brit Med Jour , 1, 485, 1947
52 YOUNG, R. H., and MCEWEN, E G. Jour Am Med Assn , 134, 1456, 1947.

anal orifice[24] The lesions may become dry, scaly, and sometimes heal completely in a few days, but usually persist several weeks, or even longer.

A more widespread skin lesion seen in 6 of our 53 cases,[20] and frequently by other observers,[17,25] is a keratotic dermatitis which appears identical with that described as "keratodermia blennorrhagicum" previously regarded only as a complication of gonococcic arthritis. The lesions have often been confused with those of pustular psoriasis[25] The patients who develop this complication are usually the most severely ill, and are quite debilitated when the keratosis begins. The lesions, most commonly seen on the soles of the feet and on the lower legs begin as flat pustules which may rupture The skin at the site of the pustules becomes crusted and horny and does not

psoriasis is marked. Some authors have described skin lesions resembling those of urticaria, erythema nodosum, and erythema multiforme. Some of these may be reactions to penicillin or other drugs. The keratosis usually lasts from one to three months, and often slows convalescence from

Summary of Clinical Features[20]

1. Acute nonspecific urethritis develops often without history of sexual exposure This may be accompanied or preceded by mild, transient diarrhea
2. Within ten days a mild but purulent conjunctivitis develops which disappears in five to ten days, regardless of treatment
3. Within two weeks after onset of discharge, an acute polyarthritis is noted, without chill, but with temperature elevation to about 101° F daily for about ten days
4. Weight loss and muscle atrophy are often marked and develop fairly rapidly
5. The urethritis clears spontaneously after three to four weeks regardless of treatment, but may recur sporadically
6. About one month after onset, superficial ulcerations may be noted on the glans penis (balanitis circinata) in many cases, and at this same time keratodermic lesions may be found on the feet and legs in some
7. The sedimentation rate is most rapid about six weeks after onset, gradually returning to normal within three months
8. The keratodermia usually clears in two months Balanitis may be recurrent independent of the existence of urethral discharge
9. Roentgenograms of the involved joints usually show osteoporosis of the approximating bone ends during the second or third month of the disease Periosteal proliferation near involved small joints is noted in some cases
10. Usually within four to six months, under supportive treatment, the involved joints appear normal, the muscles have regained their strength, all skin lesions have healed, the sedimentation rate and blood count are normal, and the changes previously seen on x-ray either have disappeared or are less marked

CHAPTER 45

TUBERCULOUS ARTHRITIS

By Victor G. Balboni, M.D.

Tuberculous arthritis is one of the most serious affections of the articular tissues. It is always a metastatic infection from tuberculosis elsewhere in the body, and from either the primary or articular focus, a fatal miliary spread of the infection may arise. The articular infection, if uncontrolled, usually proceeds slowly to complete destruction of the joint. Chronic draining tuberculous sinuses are not uncommon complications, and if these transmit secondary infection to the bones and joints amyloidosis is prone to occur. An analysis has been made of the bone and joint tuberculosis seen in a general tuberculosis hospital during fifteen years.[78] The following points were noted:

1. Three and seven-tenths per cent had single or multiple tuberculous bone and joint lesions, usually secondary to a tuberculous focus in the lungs.
2. Of those with bone and joint tuberculosis, 74 per cent had chronic pulmonary tuberculosis and 26 per cent had no pulmonary involvement
3. The frequency of joint involvement was spine—33 per cent, hip—15 per cent; fingers, toes and long bones—11 per cent, knee—8 per cent; sacro-iliac joints—8 per cent, elbow—4 per cent, shoulder—3 per cent, ankle—3 per cent, wrist—2 per cent with other regions involved in a small percentage
4. The bone and joint involvement was most prevalent in the second and third decades
5. The outcome is serious and the complications are most frequent when moderately advanced pulmonary tuberculosis is present, conservative measures should be employed in these The prognosis is also more serious when tubercle bacilli are present in the sputum, in those with a draining sinus or multiple abscesses or in those with extrapulmonary tuberculosis.
6. Almost half of the 160 patients with bone and joint lesions died, 17 per cent were well; 21 per cent were improved, and 13 per cent were unimproved.

In a study of 100 cases of bone and joint tuberculosis in individuals under sixteen years, 55 per cent of the lesions involved the spine and 21 per cent the hip. [illegible]
[illegible]
[illegible]
more sacro-iliac and knee-joint involvement.[85]

The most commonly involved vertebrae were from the sixth dorsal to the fifth lumbar. Multiple involvement was common.

the arthritis markedly. Occurrence of keratosis in Reiter's Syndrome should be regarded as a prognostic sign indicating a slow convalescence which may drag over many months.

LABORATORY FINDINGS

During the acute stage of the disease the leukocyte count averages about 12,000 per cubic millimeter. The sedimentation rate becomes elevated, but does not reach its peak for about six weeks after the onset of the arthritis, at which time the urethritis and conjunctivitis have usually disappeared, and the arthritis itself seems less acute.[19] A secondary hypochromic anemia is seen in the severe cases. Urinalysis may show clumps of pus cells, occasional red blood cells, traces of albumin, and rarely casts. Smears and cultures of exudates and fluids have shown no characteristic findings except in those cases where special cultures of urethral and prostatic secretions for pleuropneumonia-like ("L") organisms have been performed.[10]

Synovial fluid cell counts have shown from 9,000 to 53,000 leukocytes per cubic millimeter, of which 53 to 77 per cent were neutrophiles. The cell counts are therefore lower than those usually seen in arthritis due to pyogenic infections.[10] Sugar determinations on the synovial fluid in this disease have shown higher values than in pyogenic arthritides.[10] Smears and cultures of the fluid have been negative, except for growth of "L" organisms in 2 cases reported by Dienes *et al.*[10] Gonococcus complement fixation reactions on blood and synovial fluid have been consistently negative except where previous gonorrhea had occurred.[19]

Agglutination reactions of serum of patients having Reiter's Syndrome

Roentgenographic changes do not appear until weeks or even several months after the onset of the arthritis.[19] Osteoporosis of the bone ends at

ankylosis are *very rarely* encountered. Both the osteoporosis and periosteal proliferation are *apparently* reversible.[19]

DIFFERENTIAL DIAGNOSIS

Reiter's Syndrome must be diagnosed by the occurrence of the typical urinary infection, acute arthritis, conjunctivitis, and perhaps skin lesions in the absence of other specific diseases. Culture of pleuropneumonia-like organisms from the urethral exudate may prove to be a helpful diagnostic test in the future.[10] The differential diagnosis from gonorrheal arthritis is most important, as the condition is very similar clinically. Careful and repeated bacteriological studies are needed, prior to administration of chemotherapy. The gonococcus complement fixation test, when employed, has been reported negative in cases of Reiter's Syndrome unless there had

Diagnosis of Tuberculous Arthritis

Slow, insidious development *of a* monarthritis; several joints involved occasionally. About 85 per cent are monarticular.

Family history of tuberculosis, or previous personal history of *pleurisy with effusion, suppurative cervical adenitis or fistula in ano*

Associated visceral tuberculous lesion (lungs, lymph nodes) demonstrable in almost 50 per cent of cases[48]

Order of frequency of joint involvement: *spine, knee, hip,* elbow, ankle, sacro-iliac, shoulder and wrist (temporomandibular joint is rarely involved).

History of preceding trauma in 50 per cent

Symptoms: Local Presence of one or more of the following. *Doughy swelling of the joint* due to enlarged synovia rather than to fluid; *pain; stiffness; heat; limp on walking;* muscular spasm and limitation of motion; *tenderness* to palpation; muscular atrophy (sometimes early and marked). Pain and tenderness are late symptoms[21,44]

General Slight or no fever; loss of weight.

[illegible]

years of age[25]

Additional aids in diagnosis Biopsy of joint tissue; guinea pig inoculation of joint fluid; microscopic examination of joint fluid for tubercle bacilli, leucocytosis in joint fluid, decreased sugar content of joint fluid, positive tuberculin test, normal serum phosphatase.[81]

Evidence against tuberculous arthritis: Presence of considerable amount of joint fluid; arthritic involvement of four or more joints[45]

Four clinical stages have been described[79]

1. *Invasion.* physical, but no roentgen signs are present. Duration: weeks or months; occasionally several years
2. *T* [illegible]
3. *Q* [illegible] shown by serial roentgen-rays.
4. *Healing*—joint is not painful, no swelling is present. Fibrous tissue develops from tuberculous granulation tissue, fibrous ankylosis occurs Tubercle bacilli may remain for years in such fibrous tissue

With erosion of the cartilage and exposure of the ends of bones, there is increased pain and stiffness and muscular spasm, especially in sleep; this latter awakens the child with severe pain, and causing cries and jerks at night.

Progressive destruction (with remissions and exacerbations) unless properly treated.

Preservation of articular cartilage (joint space) until late in the disease.

A paravertebral abscess was found in 49 per cent of the spinal involvement in the pediatric group and in 71 per cent of the adult group At times, it was the only roentgen evidence of underlying bone disease.

[illegible] be extremely difficult to diagnose and [illegible]ethod in such cases. A tuberculous [illegible]ny persistent monarthritis and the [illegible] a careful study of the synovial fluid

onset
ondi-
The
e in-
uro-
arthritis.

The presence of urinary infection, lack of cardiac involvement except for occasional transient ECG changes,[26] negative antistreptolysin and antihyaluronidase titres, and minimal benefit from salicylate administration in Reiter's Syndrome should help in the differentiation from rheumatic fever

DIFFERENTIAL DIAGNOSIS OF INFECTIOUS URO-ARTHRITIS[30]

	Reiter's Syndrome	Gonorrheal Arthritis	Rheumatic Fever	Rheumatoid Arthritis
Age and Sex	Nearly always young males—ages 19-35	Males in ratio 3 to 1, ages 16-40	Usually ages 6-30 Either sex	Usually ages 20-40 Females in ratio 3 to 1
Family History	Nearly always absent.	Nearly always absent	Very often present.	Often present
Characteristic Onset	Urethritis usually without sexual exposure Conjunctivitis and skin lesions with arthritis.	Sexual exposure then urethritis, prostatitis and polyarthritis	Respiratory infection then fever with migratory arthritis	Usually insidious with prodromes of arthralgias, anemia, weight loss Gradually spreads.
Genitourinary Infection	Urethritis prostatitis, perhaps cystitis.	Specific urethritis and prostatitis.	None or coincidental infection	Rarely chronic prostatitis
Characteristic type of Arthritis	Acute onset, marked inflammation, polyarticular, never suppurative, tends to be asymmetrical	Very acute onset polyarticular tends to become monarticular Often suppurative	Acute onset migratory polyarticular, transient involvement	Progressive involvement usually symmetrical polyarticular, insidious spread to new joints
Characteristic Eye Lesions	Conjunctivitis (60%) Rarely iritis keratitis	Conjunctivitis (15%) iridocyclitis, ophthalmia.	Very rare	Iritis conjunctivitis—very rarely
Characteristic Skin Lesions	Balanitis circinata in 50% keratodermia in 10% mouth lesions rarely	Purpuric rash, rarely keratodermia blennorrhagica in about 3%	Erythema multiforme or marginatum seen occasionally nodules	Nodules found occasionally psoriasis occasionally
Cardiac Involvement	Electrocardiographic changes found rarely	Endocarditis rarely	EKG changes very often, endocarditis, etc	Lesions rarely found clinically
Febrile Course	Low-grade recurrent for 2-3 weeks.	Usually chill high fever with arthritis.	Usually fairly high and sustained	Occasional, low-grade remittent
Bacteriological Findings	No bacteria found except a few contaminants L" organisms often found	Gonococci from urethra often from joints	Hem strept often in pharynx High antistreptolysin titre	Indefinite Streptococcal agglutination reaction positive in over 50%
Characteristic Course	Severe in early stage self-limited, lasts 3-6 months.	Usually severe and destructive may be self-limited.	Joint involvement mild Heart lesions often severe	Progressive and destructive exacerbations and partial remissions
Response to Therapy (Chemotherapy Fever)	Not affected by sulfonamides or penicillin Streptomycin, fever sometimes effective	Responds to sulfonamides or penicillin and/or fever	Salicylates control fever, helpful to joints	No response to sulfonamides or penicillin Fever effective (?)
Sequelae and Recurrences	Residuals mild or absent. Recurrences in about 15%.	Destruction of joints common Rarely recurs.	Heart lesions persist Recurrence frequent.	Multiple deformities Chronic and progressive

The differentiation of infectious uro-arthritis from rheumatoid arthritis, particularly the more atypical acute form so often encountered in the Armed Forces during the past war, may sometimes be difficult The urinary infection closely followed by conjunctivitis and acute arthritis are certainly

and *x*-rays of the affected joint. Tubercle bacilli are often demonstrable

arthritis.
the affected
lymphaden
should be resorted to if simpler procedures leave the diagnosis in doubt.

Roentgen Findings in Tuberculous Arthritis.—The roentgen picture is not pathognomonic and may include osteoporosis, areas of rarefaction (due to abscesses) and narrowing of the joint space. Active early tuberculosis may be present in bones and joints with negative roentgen findings. Decalcification of bone and soft tissue swelling are usually the earliest roentgen signs. Later one notes marginal erosion with marked irregularities of contour along the joint surfaces, marked haziness of the joint, evidence of subchondral invasion with apparent separation of the cartilage and narrowing of the joint space

In joints with extensive contact of articular cartilage, there may occur regional atrophy of bone, decreased density of the bony articular cortex and preservation of the normal cartilage space

In joints with extensive areas of non-contact of articular cartilage (as the knee), one may see: regional atrophy of bone, narrowing or complete obliteration of the cartilage space, and decreased density of the bony articular cortex where the cartilaginous surfaces are not in contact, with preservation of bony articular cortex and cartilage space in the regions of contact (condyles and tuberosities)

If secondary invasion of the bone occurs at the traumatized points of contact, large bilateral areas of necrosis occur at opposing points in the bones, detachment of such areas forms "kissing sequestra"

Etiology.—The decrease in the incidence of tuberculous arthritis may be due to the more rigid control over dairy herds, formerly most cases of surgical tuberculosis were attributable to the bovine strain [37] Now, however, tuberculosis in man is due almost entirely to the human strain of bacilli Tuberculous arthritis may result from extension of a tuberculous process in adjacent bone, or may be a primary involvement of the synovial membrane Primary synovial tuberculous arthritis is probably more common in children than in adults Although it was formerly thought that most instances of tuberculous arthritis were secondary to tuberculosis of bone, it is often difficult to determine the site of the primary focus [73] Henderson has stated that tuberculous arthritis is always secondary to tuberculosis elsewhere in the body, primary sites include the mesenteric and bronchial lymph nodes

Pathology.—The pathology of tuberculous arthritis has been well described by various workers,[4,31 64 66] Allison and Ghormley believe that all cases of joint tuberculosis should be regarded as involvement of the synovial membrane, bone and cartilage [6]

Whether the tuberculosis is primary in the synovial membrane or in the bone there develops *a diffuse tuberculous synovitis with a pannus of granulation tissue* which tends to spread over and to destroy the articular carti-

not typical of rheumatoid arthritis [illegible] limited course of Reiter's Syndrome [illegible] as early rheumatoid arthritis may [illegible] spontaneous remissions with little [illegible] deformity [illegible] Complete differentiation of infectious uro-arthritis from [illegible] rheumatoid arthritis will await further [illegible] larly more widespread application of the [illegible] for the pleuropneumonia-like "L" organisms. [illegible] impossible to decide whether a given case was [illegible] arthritis" or Reiter's Syndrome (Infectious Uro-arthritis) [illegible] inconclusive bacteriologic study or early institution of [illegible] measures before diagnosis was made.

Pointers useful in differential diagnosis are summarized [illegible] page 807.

COURSE AND PROGNOSIS

Although practically all cases of Reiter's Syndrome [illegible] been self-limited, the duration of the attack has varied [illegible] the acute stage lasts four to six weeks. Progression of the [illegible] new joints after this period is rare, but relapses have been [illegible] arthritis had completely subsided. Recurrences of [illegible] mation, skin lesions, arthritis, and even conjunctivitis [illegible] the disease have been described.[illegible] The total duration of [illegible] varied from three to seventeen months, with the [illegible] asymptomatic in five months. In rare cases [illegible] cipitate a chronic rheumatoid arthritis, but nearly always [illegible] recovery with little, if any, joint deformity [illegible] recurrence, even after years of remission, is not [illegible] 15 per cent of our 53 cases had had previous attacks [illegible] which we treated them. In follow-up letters from [illegible] of 39 men heard from had had 1 or 2 recurrences [illegible] self-limited and producing no deformity. One [illegible] chronic arthritic involvement, of a type resembling [illegible] and another had developed an acute recurrent arthritis [illegible] the physicians in a Veterans Hospital.[illegible] All these [illegible] least two years after the admission to Ashburn General [illegible] acute uro-arthritis. The joints involved in subsequent [illegible] *same ones originally affected* in practically every case.

TREATMENT

The treatment of a self-limited disease is [illegible] treatment which requires considerable time [illegible] to have value in infectious uro-arthritis [illegible] the disease. Many treatments have been [illegible] very few warrant discussion. Since [illegible] treatment should be supportive, [illegible] possible.

General Supportive Measures.—Bed [illegible] of the disease. This is particularly [illegible]

lage.[72] In those joints in which the articular cartilages are in close con- [illegible]

[illegible]

of the cartilage. *Because of the absence of proteolytic ferments in tuberculous*



[illegible] There

exudates, this loosened cartilage may persist for months, but is finally destroyed Grooves may be formed on the articular surfaces of the bone due to invasion and absorption by the tuberculous process

Massive bony invasion at pressure points may occur in the knee and hip, producing large bilateral areas of necrosis. True osteophyte formation does not occur at the margins of the articular cartilage in tuberculous arthritis.

In one series of 224 cases, the frequency of joint involvement was as follows: spine, 49 per cent; knees, 20 per cent; hips, 11 per cent; sacroiliacs,

weight-bearing joints. In the milder cases, partial bed rest and restriction of activity may be sufficient. The period of bed rest should not be prolonged beyond the time of the febrile reaction, as muscle wasting often develops rapidly and may slow convalescence considerably.[20] Bed *exercises* at frequent intervals should be prescribed for those patients confined to bed to prevent muscle atrophy and contractures. Involved joints should be rested, but not necessarily splinted.

The *diet* should be adequate, high in total calories and vitamins A high protein intake should help bring the nitrogen balance back to the positive side It has been shown[16] that a negative nitrogen balance develops with any acute disease and that exercise and adequate protein intake can cut short this period of nitrogen loss from muscle breakdown.

Physical therapy has an important place in the treatment of Reiter's Syndrome. When joints are acutely painful, the application of surface heat, particularly hot packs, is quite soothing Hydrotherapy in a tub or whirlpool bath, when available, is extremely helpful in preserving normal muscle tone. Since permanent deformities seldom result from infectious uro-arthritis, this therapy is most cases need not be prolonged, and improvised home measures are often quite successful in relieving the patient's symptoms

Local Symptomatic Treatment.—For treatment of the *urethritis*, probably the most soothing and helpful measure is occasional urethral irrigation, using a 5 per cent mild silver proteinate or a similar bland solution [6] When posterior [illegible]
times dail [illegible]
be scrupu [illegible]
quently follows such procedures. The *conjunctivitis* also may be controlled by compresses or bland irrigations of mildly antiseptic solutions [27] Boric acid or bland silver preparations will ease the troublesome irritation Local use of chemotherapeutic measures is not recommended. In rare instances, the *diarrhea* may be severe enough to require administration of paregoric or other opiate. Bismuth or kaolin preparations usually suffice The *skin lesions* have been managed in many different ways.[17,25] Probably as satisfactory as any has been local inunction of 5 per cent ammoniated mercury ointment, and daily soaks or wet packs using dilute (1 to 8,000) potassium [illegible]

in Reiter's Syndrome, but results are poor These agents may be employed as a therapeutic test If the patient responds promptly to such treatment the diagnosis is immediately thrown into doubt.

Fever Therapy, by intravenous injection of typhoid vaccine or other pyrogen has been credited with some success in treatment of Reiter's Syndrome.[2,17,26,32,42,44,45] The course usually consists of 6 to 8 bouts of fever from the increasing doses of vaccine given every other day. Patients with this disease are seldom so debilitated that this form of treatment would be dangerous. The use of *gold salts* in the treatment of Reiter's Syndrome has been successful in only a few cases[51] In 2 of our cases the disease was aggravated by this therapy.[21] If the arthritis of Reiter's Syndrome is prolonged and severe, a course of gold therapy might be justified if other

7 per cent; tarsus, 6 per cent; ankles, elbows, wrists, each 2 per cent; shoulders and symphysis pubis, under 1 per cent.[15] Involvement of an ankle, knee, elbow and spine has been reported in the same individual.[80]

Although the tuberculous process may be quiescent, it rarely heals. If secondary sequestra are present and the process remains active, bony ankylosis almost never occurs although fibrous ankylosis may be noted when the articular cartilage is completely destroyed.

PRINCIPLES OF TREATMENT

The first step in treatment of a suspected case of tuberculous arthritis is to put the patient on bed rest, preferably in a hospital, and to completely immobilize the suspected joint while the necessary diagnostic studies previously described are performed. Here again it is emphasized that tuberculous arthritis is always a metastatic infection, so great care must be taken in seeking out the primary focus and other possible metastatic lesions Once the diagnosis is made, the responsibility of treatment should be shared equally by the internist or rheumatologist and orthopedic surgeon. The internist should institute a program designed to arrest the infection not only in the joint but also the primary focus if it is still active This program will include: (1) complete immobilization of the joint, (2) a high vitamin, high caloric diet; (3) strict bed rest if a weight-bearing joint is involved or if multiple foci exist (complete bed rest may not be necessary if the disease is localized to a joint in the upper extremities), (4) heliotherapy; (5) antibiotics and chemotherapy as described in the following section. The orthopedist makes the necessary splints to immobilize the joint and then when the infection is arrested, solidly fuses the joint so that permanent healing can take place. Occasionally in young patients when

and full motion without pain These criteria are rarely attainable even with the best of medical programs including streptomycin, PAS, and isonicotinic acid hydrazide Fusion is customarily carried out when the

tion about joint by x-ray. Tuberculosis heals very slowly, if ever, in elderly patients and here amputation of the affected limb is often the most practical form of therapy. Girdlestone and Somerville[92] in the recent edition of their book on Tuberculosis of Bone and Joint, summarize the situation as follows· "Children heal better than adolescents, adults heal slowly, and the elderly never achieve a final arrest In children, conserva-

heal

treat-

adults

when it can be foreseen that, without operation, healing will be delayed, uncertain, and probably unsound The need for operation is often obvious,

not typical of rheumatoid arthritis as we have known it. Even the self-limited course of Reiter's Syndrome may not be a definite differential point, as early rheumatoid arthritis may have rather marked exacerbations and spontaneous remissions with little residual deformity in some cases. Complete differentiation of infectious uro-arthritis from "atypical" acute rheumatoid arthritis will await further long-range case study, and particularly more widespread application of the techniques for bacteriologic study

page 807.

COURSE AND PROGNOSIS

Although practically all cases of Reiter's Syndrome so far reported have been self-limited, the duration of the attack has varied widely. As a rule, the acute stage lasts four to six weeks. Progression of the inflammation to new joints after this period is rare, but relapses have been seen before the arthritis had completely subsided. Recurrences of genito-urinary inflammation, skin lesions, arthritis, and even conjunctivitis during the course of the disease have been described.[19,26] The total duration of an attack has varied from three to seventeen months, with the usual case becoming asymptomatic in five months. In rare cases[21,44,45] the infection may precipitate a chronic rheumatoid arthritis, but nearly always the prognosis for recovery with little,
recurrence, even after
15 per cent of our 53
which we treated the
of 39 men heard from had had 1 or 2 recurrences of the uro-arthritis, again self-limited and producing no deformity. One more of these men had a

least two years after the admission to Ashburn General Hospital for the acute uro-arthritis. The joints involved in subsequent attacks were the *same ones originally affected* in practically every case.

TREATMENT

The treatment of a self-limited disease is very difficult to evaluate. Any treatment which requires considerable time for its administration will seem to have value in infectious uro-arthritis because of the natural course of the disease. Many treatments have been tried in Reiter's Syndrome, but very few warrant discussion. Since the disease is usually self-limited, treatment should be supportive, symptomatic and conservative as far as possible.

General Supportive Measures.—Bed rest is essential in the acute stage of the disease. This is particularly necessary when the arthritis involves

from the first. In an adult, for example, it is no good whatever hoping for the natural restoration of movement in a tuberculous hip, knee or ankle. In some joints the infected tissues can be excised, in others, an extra-articular synostosis can be brought about. Provided this removes all strain from the joint, the patient can be allowed up as soon as his general condition will benefit thereby. We can then accept the principle that operation will accelerate sound healing and often bring it about when it would not otherwise take place; but we cannot afford to forget two vital principles: (1) that operation on a joint has no direct influence on deep-seated lymphatic tuberculosis, and (2) *that operation will be far more certain of success if it is done at a stage when the general condition of the patient is at its best and the local lesion is no longer active.* One ought then in general to

For example, one may feel it advisable to perform thorough excision of the focus in the hip, or excision of the knee in older patients, in order to give relief from a massive tuberculous focus. One should not hesitate to amputate the leg of a man over 40 with a widespread active tuberculosis of the tarsus."[92]

ANTIBIOTICS AND CHEMOTHERAPY

Streptomycin.[92,93,94]—More than four years of experience has shown that streptomycin is a really effective drug in the struggle against tuberculosis

rapidly and to a greater extent than formerly, and surgical procedures made possible at a much earlier date *The currently accepted dose is 1 gram, intramuscularly, every third day for four months and twelve grams PAS daily concomitantly* Toxic manifestations rarely develop on this dosage and tubercle resistance develops less rapidly with intermittent therapy of this type. Some investigators prefer 1 gram daily but there is no evidence that this is more effective than 1 gram every three days.

Dihydrostreptomycin.[93]—This modification was originally thought to be less toxic than the original drug. Now, however, its special value is

ve

o-

mycin and tends to delay the emergence of resistant forms of tubercle bacilli The drug is given by mouth in 3 gram doses, four times a day. ... but whenever

... cotinic acid all show a bactericidal effect in vitro against mycobacteria tuberculosis. The original clinical reports were most optimistic, but as further reports have come in, it appears that these drugs are about as effective, or possibly slightly less so, than is streptomycin. Bacterial resistance, which at first was thought not to occur, has now been shown. These drugs are still in

within two weeks without recurrence, and the third case seemed to subside
d an exacerbation
Further studies
e evaluated criti-
cally, but streptomycin therapy is certainly worthy of trial in infectious uro-arthritis[21,49] Brown *et al.*[5] have found that most human strains of

In treating Reiter's Syndrome, therefore, streptomycin and terramycin

in each was prompt and dramatic, but the symptoms returned in all three

withdrawn, and appeared chronic From these findings it appears that the hormones cause a prompt suppression of symptoms while being administered, but have little or no effect on the cause or natural course of this usually self-limited disease Certainly the use of ACTH, cortisone, or hydrocortisone should not be recommended for treatment of Reiter's Syndrome except when antibiotics have failed

CONCLUSIONS

There has been much controversy and disagreement concerning Reiter's Syndrome. This has stemmed largely from early conflicting reports[12 31] of cases inadequately studied, and from insistence on the part of some that Reiter's Syndrome must consist of urethritis, conjunctivitis, and arthritis, of others that the syndrome is variable, and of still others that any non-gonococcal urethritis with arthritis represents the Syndrome

This self-limited form of arthritis, with a rather spectacular clinical pattern, has been confused with many other ill-defined diseases, but the development of methods for isolating the etiologic agent, and greater usage of these methods, should help clarify the present confusion If streptomycin or terramycin therapy indeed represent a specific treatment for the infection, *a valuable therapeutic tool, as well as diagnostic test,* will have been added The subsidence of symptoms and signs on ACTH or cortisone administration does not augment our knowledge, since this is a non-specific suppression of the reaction without effect on the causative agent Further careful study is needed

BIBLIOGRAPHY

1 BAUER, W, and ENGELMAN, E P Trans Assoc Am. Physicians, 57, 37, 1942
2 BEIGLBOCK, W Deutsche med Wchnschr, 69, 803, 1943
3 BEVERIDGE, W I B Med Jour Australia, 2, 479, 1943
4 BEVERIDGE, W I B, CAMPBELL, A D, and LIND, P E Med Jour Australia, 1. 179, 1946

clude central nervous system stimulation (hyperactive reflexes, twitching, insomnia and bladder and sphincter disturbances Hemopoietic disturbances have been seen in animals but not so far in man. These drugs should

tuberculous sinuses

TUBERCULOSIS OF THE HIP JOINT

The *primary lesions* are usually in the *neck or head of the femur*, these may result in complete disappearance of the head of the femur and destruction of the acetabulum. Other important points include:[33]

Early synovial invasion
Marked rarefaction of bone with obliteration of landmarks
Misshapen or entirely absorbed femoral head
Occurrence in children, in most cases, under the age of ten

Early symptoms: *Limp pain in the knee and thigh* from pressure on the obturator and anterior crural nerves, *muscle spasm, fullness in the groin, tenderness on pressure over the hip;* slight flexion and abduction of the thigh; flat
the affect
and abdu
abscesses
of the thigh.

Treatment.—Combined streptomycin and chemotherapy, rest, high

secure separation of the diseased surfaces. Months or years are required for fusion which is the ideal treatment. Motion in the hip joint will result in relapses and deformity The procedure of choice in the hip, even in

18 per cent.[19] It was noted that *the outlook was more favorable if* the arthrodesis was not done before eight years of age or after thirty years, if ther was no active pulmonary tuberculosis or sinus or cold abscess abou

measures have failed.
of rheumatoid arthritis.
has had a definite curat
somewhat toxic drug in a self-limited process unless the disease is very severe or prolonged.

Streptomycin.—In the arthritis of rats infected with pleuropneumonia-like ("L") organisms, Powell, Jamison, and Rice[34] found that streptomycin given parenterally was quite effective in terminating the joint infection Deines *et al.*[10] used streptomycin (2 to 4 gm. daily) in 8 patients with "L" organism infections of the genito-urinary tract and in five patients with Reiter's Syndrome. In 7 of the 8 cases of genito-urinary infection, there was a rapid disappearance of symptoms during treatment and cultures became negative. In Reiter's Syndrome there was an improvement of symptoms during and after streptomycin therapy, but in most of these cases the joint inflammation itself persisted for some time and streptomycin did not prevent recurrences of the disease. Three of our cases of Reiter's Syndrome at the Hospital of the University of Pennsylvania were treated with streptomycin in doses of 2 grams daily for one week.[21] There was marked improvement of all symptoms in one case with recurrence three weeks after discontinuation of treatment One case subsided completely

Summary of Treatment of Reiter's Syndrome[20]

Although the prognosis for this condition is good, several important
patients.
tried, but *only* after
ruled out gonorrheal
infection Many cases are confused by inadequate study and eager institution of chemotherapy. If the disease responds to chemotherapy with either penicillin or sulfonamides, the diagnosis of Reiter's syndrome (infectious uro-arthritis) becomes doubtful

2 Salicylates, in doses of 4 to 6 gm. daily, are helpful in controlling joint pain

3

residual disability.

4. Symptomatic therapy for the eye and skin lesions are perhaps helpful, as are bland urethral irrigations and hot sitz baths for the genitourinary infections, but vigorous therapy may be harmful, prolonging the natural self-limited course of these infections.

5 When the diagnosis has been established, the patients should be reassured concerning prognosis.

6 A course of fever therapy by intravenous injection of typhoid vaccine may be attempted in the most severe or persistent cases This has seemed to hasten recovery in some of our cases

7 Streptomycin therapy (2 to 4 gm. daily), or terramycin (1 to 2 gm daily), should be tried for 4 to 7 days.

8 In severely ill patients, or where above measures have failed, the administration of ACTH (40 to 60 mg /day), or cortisone (75 to 100 mg /day) is recommended.

hip at the time of operation and if there was no secondary pyogenic infection about the hip.

TUBERCULOSIS OF THE KNEE JOINT

Tuberculosis of the knee is a *common type of joint tuberculosis in adults* It occurs in 11 per cent of all tuberculous bone and joint lesions. Synovial tuberculosis progresses to bony changes in 45 per cent of cases.[84] *Tuberculous arthritis of a low grade may persist in a knee for years with very little discomfort. Local signs include* heat, swelling, deformity, stiffness, pain, limping, muscular spasm, restriction of motion, and occasionally an intra-articular effusion. Flexion deformity of the knee and contraction of the hamstring muscles may develop. Slight jarring of the joint may produce excruciating pain.

Early diagnosis of tuberculous arthritis is aided by use of synovial membrane biopsy (which may show tubercles in as high as 82 per cent of cases) and aspiration of joint fluid for special cultural methods of tuberculosis and guinea pig tests

Key summarized his findings in *adults with tuberculosis of the knee joint.*[81]

6 per cent had abscess formation at an early stage

arthritis, gonorrheal arthritis, syphilis, hemophilia, traumatic lesions and neoplasm.

At present, most individuals treat tuberculous arthritis in children conservatively for a time, but treat adults surgically unless an upper limb is involved—when one may temporize for a while. *Tuberculous arthritis is a serious disease with a high mortality* Although operation alone will not cure the individual, it may turn the tide in his favor.[16]

Ober reported some cases of tuberculosis of the knee in which complete recovery occurred without operation, but pointed out that in these cases the tuberculous process did not extend into the knee joint or through the epiphysis, but extended laterally and decompressed itself through the side of the bone medially or [illegible]. Fusion is not do[illegible] patients [illegible]

operation Immobilization in a brace or cast for months or years has been used in children, with the knee in complete extension to prevent flexion deformities Results of conservative treatment in children are usually not good.

In individuals from young adult life to middle age, orthopedic surgeons are agreed that the treatment of choice is surgery to produce femorotibial synostosis. In the aged, tuberculosis of the knee joint is usually treated (in most instances) by *amputation* at the thigh. It is possible now with prolonged use of newer chemotherapeutic measures such as streptomycin and PAS to arrest some tuberculous processes that are limited to the synovia or periarticular tissues However, if bone or cartilage is involved, surgical fusion is indicated as soon as the process is controlled by all other measures

There is still a *lack of general agreement as to the advisability of surgical measures in treatment during the years of growth* while epiphyseal cartilages are still present. Most surgeons prefer not to resect the knee in individuals during the period from birth to epiphyseal closure because some have claimed that. (1) tuberculosis of the knee heals under conservative treatment in the child, and (2) that the resulting deformity and shortening following resection in the child are too crippling to justify this procedure [63]

Following resection, the knee joint of a child ankyloses very readily and the percentage of successful fusions following a single operation is high In one series, marked shortening occurred in only 30 per cent of the infants and children treated [59] Other reports[27 39 41 86] also seem fairly optimistic concerning results of operative management Short wave therapy,[84] fever therapy[26 71] and gold[30] are of very little or no value in treatment. Streptomycin and PAS (para-amino salicylic acid) greatly shorten the pre- and post-operative periods (see section on Antibiotics and Chemotherapy), and should be given a trial in all cases in conjunction with the standard conservative procedures

TUBERCULOSIS OF THE SPINE

(Pott's Disease; Tuberculous Spondylitis)

This is seen most often between the ages of two and ten It is the *most common form of tuberculous arthritis.* Trauma may be a predisposing factor in this disease.[81]

Approximately 1 per cent of all deaths from tuberculosis in the United States are due to Pott's disease [6] Pott's disease is considered to be a local manifestation of a chronic general tuberculous infection [50]

The lesions occur most commonly in the bodies of the vertebræ although the intervertebral disk may be affected first,[13] in more than one-half of these cases, the thoracic vertebræ are affected The anterior border of the [illegible]

weight bearing

Isolated tuberculosis of a vertebral spinous process has been reported

Cold abscess formation and *paralysis* are the two *commonest complications* Such an abscess in the cervical region may be found laterally in the neck

SECONDARY STAGE OF ACQUIRED SYPHILIS

Bone and joint syphilis is rare in the secondary stage of the disease and presents no characteristic signs or symptoms.[3] Vague aches in muscles or bones—*arthralgia*—occur in about 5 or 10 per cent of patients in the early stage of acquired syphilis; these are most common during the height of the systemic reaction. They are usually more of an *ache* than a severe pain, are often *nocturnal*, and are not increased by gentle motion of the joints.

Syphilitic Arthritis.— [illegible] of acquired syphilis.[19] Despite this, syphilitic [illegible] entity by the ancients and to have been well described in the Middle Ages. Efficiently treated cases of syphilis do not develop specific arthritis.

Syphilitic arthritis usually involves *multiple joints* and is seen most frequently in the *large joints* (especially the knees);[17 18] involvement of the sterno-clavicular joints may occur.[14] An acute or chronic synovitis may be seen, this is *frequently bilateral*. The joints may appear swollen and may show a fusiform appearance, with thickening of the synovial membrane and surrounding tissues and some muscular spasm and atrophy. In other cases, there may be no swelling of the joints. There is very little limitation of motion, local heat or muscular spasm. Muscular wasting is usually of minimal degree.[18] The physical and roentgen manifestations are usually far greater than the pain, muscular spasm and disability. Although bony ankylosis does not occur, impairment of function and deformity may be found. The Wassermann reaction is positive in most cases.

The disease usually responds promptly to efficient antisyphilitic therapy, but not to salicylates.

Hydrarthrosis.—Transient hydrops may occur in early secondary syphilis; this may be polyarticular but usually affects *the knees*. An abundance of fluid is present. There is a moderate amount of pain but usually no redness of the joint. Treatment with arsenicals or penicillin is usually effective. In some cases, a more persistent form of hydrarthrosis may occur with slight disability and very little muscular wasting.

Tenosynovitis.—This is usually a *mild, symmetrical, painless* swelling of tendon sheaths, most often occurring bilaterally.[42] Infiltration of the tendon sheath may occur in the later stages.

Bursitis.—Syphilitic bursitis is usually painless or only slightly painful, there is swelling, bursal effusion and a tendency to break down with sinus formation. It may also occur during the tertiary stage.

Syphilitic Periostitis.—This does not usually cause joint pain, the [illegible] ent to a [illegible]

[illegible] invasion [illegible] spondyl- [illegible] osteitis [illegible] affects chiefly those bones exposed to trauma and pressure. Syphilitic spondylitis may occur in congenital syphilis or in acquired syphilis.

Symptoms of syphilitic spondylitis include localized pain which may be more marked at night, stiffness, tenderness to palpation and avoidance

hip at the time of operation and if there was no secondary pyogenic infection about the hip.

TUBERCULOSIS OF THE KNEE JOINT

Tuberculosis of the knee is a *common type of joint tuberculosis in adults.* It occurs in 11 per cent of all tuberculous bone and joint lesions. Synovial tuberculosis progresses to bony changes in 45 per cent of cases.[64] *Tuberculous arthritis of a low grade may persist in a knee for years with very little discomfort. Local signs include* heat, swelling, deformity, stiffness, pain, limping, muscular spasm, restriction of motion, and occasionally an intraarticular effusion. Flexion deformity of the knee and contraction of the hamstring muscles may develop. *Slight jarring of the joint may produce* excruciating pain.

Early diagnosis of tuberculous arthritis is aided by use of synovial membrane biopsy (which may show tubercles in as high as 82 per cent of cases) and aspiration of joint fluid for special cultural methods of tuberculosis and guinea pig tests.

Key summarized his findings in *adults with tuberculosis of the knee joint*[51] *Pain* was the first symptom in 70 per cent of cases; this was usually insidious in onset. In 22 per cent of cases, *swelling* of the knee was the only initial symptom *Stiffness* was the initial symptom in 10 per cent. *Local heat* was always present and there was usually *gross limitation of motion.* About 6 per cent had abscess formation at an early stage.

In 87 per cent, the patient's general physical condition was good. Diagnosis of early cases was often extremely difficult; the *most valuable laboratory aid* was bacterial examination of the pus from an abscess (including guinea pig inoculation.) *This disease may be confused in adults with* rheumatoid arthritis, gonorrheal arthritis, syphilis, hemophilia, traumatic lesions and neoplasm.

At present, most individuals treat tuberculous arthritis in children *conservatively for a time, but treat adults surgically unless an upper limb* is involved—when one may temporize for a while. *Tuberculous arthritis is a serious disease with a high mortality.* Although operation alone will not cure the individual, it may turn the tide in his favor.[16]

Ober reported some cases of tuberculosis of the knee in which complete recovery occurred without operation, but pointed out that in these cases the tuberculous process did not extend into the knee joint or through the epiphysis, but extended laterally and decompressed itself through the side of the bone medially or laterally Fusion is not done in these patients as healing occurs without any disturbance to the knee joint

Ober believes that *operation on a tuberculous joint is contraindicated if there is any indication of active pulmonary tuberculosis; a chest x-ray should be taken in all patients with tuberculosis of joints.*[67]

Attention must be paid to the *general treatment of the patient* and not only to his affected joints Rest of the joint with splints, extension by weights and rest in bed is usually the treatment of choice in children. *In adults, conservative treatment has been abandoned because the results are poor.* If the knee is involved, *resection of the affected articular ends of bone with bony ankylosis of the remaining parts is the ideal treatment in adults.* The patient can often return to work within a year following such an

in the early months of congenital syphilis.[21,24] This occurs as an *osteochondritis in a juxta-epiphyseal* location, which is at times accompanied by considerable periarticular swelling. It is seen in approximately 5 per cent of children with congenital syphilis.

It is most common in the first three weeks of life and rarely begins after three month
the lower.
cartilage wh
epiphyseal separation (Parrot's pseudoparalysis) or fracture of the epiphysis may occur.

On roentgen examination, one sees widening of the joint space, and irregular epiphyseal line, periosteal thickening, decalcification of bone just proximal to the cartilage, cupping of the diaphysis and an irregular *density and streakiness of bone adjacent to the cartilage.*[8,12,25]

If proper treatment is given in time, the joint may undergo complete resolution; shortening or deformity will occur if the growing line has been *greatly damaged* [20]

Clutton's Joints (Symmetrical Synovitis in Congenital Syphilis).—Clutton first described a symmetrical synovitis of the knees in congenital syphilis in 1886.[8] This is *one of the most common forms of syphilis of the joints.* Clutton's joints occur in congenital syphilis, usually appearing *between the ages of eight and sixteen.* They are characterized by a *marked painless symmetrical swelling* of the periarticular tissues with effusion into the joints.[26] This develops insidiously.

The involvement is usually limited to the soft tissues. The swelling produces some mechanical impairment to motion. The chief sites of involvement are the *knees* and elbows; the knees are involved in about 85 per cent of the cases. These patients are able to walk well despite the effusion. There is usually *no fever* and there is *very little local heat, pain or tenderness* Interstitial keratitis is often an associated finding

The *fluid reaccumulates rapidly if aspirated* and may persist for years without permanent damage to articular tissues.[28] From 10,000 to 45,000 leukocytes (chiefly lymphocytes) have been found per c mm. of this fluid. A similar form of synovitis may occur in early or late acquired syphilis Polyarticular joint involvement has also been reported [6,7]

Roentgen examination is usually negative except for the separation of ... always ... ildren [46] ... *well to*

... hildren and is uncommon. ... *or toes in children from* ... [22] A characteristic spindle ... nities and marked shortening; *in some cases, it leads to absorption of bone*

Miscellaneous.—Gummata of bone, Charcot joints and spondylitis are uncommon manifestations of late congenital syphilis. Gummatous lesions are rare under twelve months of age.[20] In congenital syphilis, one may see joint lesions which are identical with those of the tertiary stage of the acquired form. Aseptic necrosis may occur in congenital syphilis, resulting in the formation of "pus" within the joints.

Mayer reported that if operation was performed during active tuberculosis, fusion did not alter the course of the tuberculous process, but that if it was done during the reparative stage, it was beneficial in that it strengthened a weak spinal column *In most reported series, mortality rates are lower among patients who have been operated upon than in those treated without operation.* Meyerding preferred conservative treatment for



Fig 257 —Tuberculosis of the spine showing calcification in a small psoas abscess (Holmes and Robbins)

children but noted that results following fusion operations were better for adults [60]

Paralysis of the legs complicating Pott's disease usually results in recovery within several months following the use of a convex frame.

Finkelstein made a careful ten year study to determine whether fusion operations shortened the course of tuberculous spondylitis or whether

impairment of motion Although such joints may be large and tense, there is *very little evidence of inflammation*, muscular spasm, wasting, limitation of movement, constitutional reaction or ankylosis (unless secondary infection occurs).

Gummatous deposits may be found in perisynovial tissues and may occur in the cancellous ends of the bone and later spread to cartilage.[29, 32] Although the bony changes are most marked, all of the joint structures are usually involved in the process In young individuals, the process may begin as an epiphysitis. One may see erosion and irregularity of the joint surfaces, cavitation within the bones and bony outgrowths. In adults, the condition may resemble degenerative joint disease When the spine is the picture simulates that of tuberculosis.



In adults, one large joint is usually affected Extensive bony changes may be seen; these may be confined to the joint or may extend the whole length of the bone. Osteophytes are common Gumma of the patella has been reported.[2] If specific therapy is not instituted, the joint may become ankylosed. **Syphilis and tuberculosis should always be suspected in a monarthritis or deforming arthritis at any age**

Juxta-articular Gummata.—These may occur in tertiary syphilis and lead to symptoms in the joint.[16] Roentgenogram may show considerable

Fresh air, heliotherapy and a high caloric diet are essential in these youngsters. Streptomycin and PAS should be used routinely in all cases.

Ambulatory treatment may be begun after several months of freedom from symptoms. A plaster jacket is used for support when the patient is out of bed. Spinal support must always be used when the patient is up. When ambulatory, a light brace may be used for spinal support and for prevention of dangerous movements of the spine.

Harris and Coulthard studied the bone and joint lesions of 296 patients who were followed for a period of from one to ten years. Only 8 per cent of these were in children (as their hospital accommodated chiefly adults). Their *best results were obtained by* treating bone and joint tuberculosis first by *rest* and controlling other tuberculous foci in the body; *then, by operation to obtain bony ankylosis* of the affected joint. Fibrous ankylosis or unstable joints often marked the end result of non-surgical treatment; the results of those with bony ankylosis were superior to those treated non-surgically.

In 88 per cent of patients, tuberculosis of the spine was arrested by spine bone graft.[43]

Over 40 per cent of the patients had *pulmonary tuberculosis; this is the most serious of the tuberculous complications.* Prognosis is affected favorably by the adequate treatment of abscesses (to prevent secondary infection of bone) and by freedom from multiple tuberculous lesions in the body. Secondary infection of the spine or of the sacro-iliac joint or hip rendered the prognosis poor. Secondary infection of the knee, ankle or foot required excision or amputation. However, today modern antibiotics are proving of great help in eradicating or controlling secondary infection.

About a third of the patients were free from multiple tuberculous foci

Treatment of tuberculous spondylitis must include relief of pain; healing of the spinal lesion; prevention or cure of deformity, of complications and of relapses as well as prolongation of life.[36]

Prolonged general care is always advisable in Pott's disease; surgical fusion should be postponed until the resistance of the individual is increased and until the lesion becomes stationary or begins to heal.[14,58,60] Surgical fusion of the vertebræ should be considered only as an aid and not the cure of Pott's disease

When active tuberculosis is localized to the spinal column, the mortality rate in Pott's disease may be less than 10 per cent;[17] if active tuberculosis is present elsewhere in the body, the mortality may surpass 50 per cent It is evident that *fusion operations should not be attempted until tuberculosis of the lungs or other organs is controlled.*

In older children and in adults, arthrodesis is advised in order to fuse the bodies of the vertebræ Unfortunately, it is not possible in this case to remove the diseased bone. In adults, following spinal fusion, an apparatus to support the spine should be worn for at least a year Haas favors early spinal fusion even in children.[38]

Spontaneous healing of tuberculous spondylitis is rarely seen.

Abscesses complicating Pott's disease may be treated as follows:

[illegible]

sary.

ring of the

wound to prevent sinus formation.

only sees coëxisting lesions of the sternum,

by: (1) evidence of active syphilis, (2) marked improvement under antisyphilitic therapy; and (3) suggestive *x*-ray findings. The disease may be limited to only one vertebra or may involve several adjacent vertebræ. One may see destruction of bone or new bone production, or a combination of the two. In most instances, more or less spur formation occurs around the vertebræ simulating the appearance of a non-specific inflammatory spondylitis

Freedman and Meschan reported 3 cases of syphilitic spondylitis with substantiation by necropsy findings in one.[16]

by

in

wrists or elbows. In children

Keratitis is present in about three-fourths of these patients.

In *young adults*, there may be involvement of several joints in succession with tender, swollen, red painful joints and with fever of 103° F. or more. Marked sweating and anemia may occur. Salicylates do not result in improvement. In most instances, the Wassermann reaction is strongly positive. Treatment with

Rheumatoid Form of Syphilitic Arthritis.—Very rarely, syphilis of the

diagnose syphilitic arthritis merely on the basis of a positive serology in a patient with joint disease.[19]

TERTIARY SYPHILIS

the

se tous

ar

the

early stages of tertiary syphilis but may also occur in children with congenital syphilis. Larger joints are affected most frequently. Considerable amounts of painless effusion are characteristic of the process; occasionally severe pain may occur. Other features include the presence of an irregular lumpy, localized swelling about the joint with very little restriction of motion or muscular wasting. The gummatous process may affect the whole joint or may remain localized. This may resemble tuberculosis of the joint. In children, these have been known as *van Gies' joints*. In severe gummatous arthritis in tertiary syphilis, suppuration may be the end result.

Osseous Form of Gummatous Arthritis.—This usually involves large joints. Extensive gummatous involvement may occur with very little

...tion in a tuberculous joint does not produce tuberculous meningitis [56]

Although arthrodesis is generally considered to be the treatment of choice in tuberculous joints, recovery with function has been reported [22]

TUBERCULOUS TENOSYNOVITIS

This condition is rare, occurring only 36 times in the past forty-five years at a large general hospital.[3] The average age incidence was thirty-six years (ranging from fifteen to seventy-five years). The right hand was involved in 22 cases (volar surface in 15, dorsal in 6 and both in 1) and the left hand in 14 cases (volar = 8, dorsal = 5 and both = 1) None of the patients were laborers, although 30 made hard use of their hands Thirteen of the individuals had tuberculous lesions elsewhere in the body

The diagnosis was made by the finding of a gradually developing painless or slightly tender mass (most often on the volar aspect of the hand), with partial stiffness and inability to completely flex or extend the fingers Grasping strength ...

Treatment consists of rest by splinting, and if necessary, incision and drainage or resection of the involved tendon sheath Streptomycin and PAS should be used, and continued until all evidence of activity has subsided.

TUBERCULOUS RHEUMATISM

This is the name applied by various observers, especially in Europe, to a type of polyarticular joint involvement, simulating rheumatoid arthritis, which may be of tuberculous etiology, but in which the tubercle bacilli cannot be demonstrated in the joints by smear or culture [18 19 21 34] The existence of such an entity is doubted by many individuals in this country. Most American rheumatologists believe that *this condition is merely the occurrence of rheumatoid arthritis in an individual who happens to have an associated visceral tuberculous lesion* [35]

The clinical picture of tuberculous rheumatism was described by Poncet in 1897; it is now known by some as **Poncet's disease** [74–76] Some individuals believe that *tuberculous rheumatism may be caused by* (a) *toxins from* tubercle bacilli at a distant focus; (b) attenuated tubercle bacilli in the joints, (c) hypersensitivity (allergy) to primary evanescent tuberculous or other foci;[34] (d) an ultravirus of tuberculosis not yet demonstrable microscopically, or (e) tuberculous involvement of the nervous system with ...

h...

m... arthritis, and to various experimental, laboratory, therapeutic and roentgenographic evidence. The excellent résumé of this subject by *Bray* and *Hench* gives in detail the pros and cons of this argument [11] These investigators *can find no incontrovertible proof to support the existence of*

NEUROGENIC ARTHROPATHY (CHARCOT JOINTS)

Mitchell first referred to tabetic arthropathies in 1831.[35] Charcot described these joints in 1868[4] About 5 or 10 per cent of tabetic patients and 25 per cent of those with syringomyelia develop arthropathies, they are much more frequent in males than in females. *Charcot joints may occur* in tabes dorsalis, syringomyelia[11] and occasionally in paresis, diabetes, leprosy, poliomyelitis, compression and destruction of areas of the spinal cord or following peripheral nerve section.[41] Although Charcot joints begin most commonly after forty years of age, they may occur as early as the age of twenty. The disease may be produced in animals by section of the posterior (sensory) roots [13]

Kernwein and Lyon commented upon the three *theories advanced for neuropathic joints*· traumatic, trophic and the mechanical [23] In numerous individuals with classical tabes dorsalis (including ataxia, hypotonia and loss of sensibility deep in the joint) neuropathic joints never develop Only unilateral neuropathy may develop in others with bilateral sensory changes, in these, a history of trauma can usually be elicited, the severity of the injury being overlooked because there is no pain It is apparent that loss of articular sensation alone does not cause the development of a neuropathic joint

Various observers have shown repeatedly that neuropathic joints may be produced in dogs, cats or rabbits by destroying the articular sensation by cutting the posterior roots In a case reported by Kernwein and Lyon severance of the sciatic nerve was followed by atrophic changes in the muscles of the leg, the ankle became relaxed because of a loss of motor nerve supply. There was an ataxia and anesthesia below the knee The trauma associated with walking on such a limb was frequently excessive because of the absence of the protective sense of pain A neuropathic arthropathy of the ankle developed with disintegration of the joint Roentgen examination showed changes typical of neuropathic arthropathy

Loss of deep sensation with an added factor of *acute or chronic trauma* in some cases leads to *effusion, relaxation of ligaments, chronic strain* [illegible] ondensa- ractures ular and ligaments [illegible] the joint – the end lar in either tabetic ter they are more and do not often go on to the extreme destruction as is seen in the lower extremity with tabes dorsalis (probably because weight-bearing joints are not affected) Charcot joints have been reported in the hip,[10] knee, shoulder, elbow, hand, foot, ankle, spine and [illegible]

bet [illegible]
tne [illegible]
gin [illegible]

spontaneous fractures. Spontaneous transverse fractures are most commonly seen in the femur, tibia and forearm, but may also occur in the tarsal bones.

such operations prevented deformity.[29] Twenty-six patients had Albee type fusion operations while 17 others of similar age group, severity and situation of disease were treated by conservative measures only (rest on frames, sunlight, good food, fresh air, etc.). *The non-surgical group was cured in 25 per cent less time* than the surgical group so that the operation in their hands seemed to prolong, rather than shorten the course of the disease. The disease in the vertebræ seemed uninfluenced despite firm fusion. *Approximately the same number of recurrences, spinal deformities, abscesses and paraplegias developed in the conservatively treated and the surgically treated groups.*

Swett and his associates discussed the end results in 350 cases of Pott's disease, evaluating the *merits of operative and non-operative treatment* [82] They found that *healing rarely occurred when an abscess was present*, especially if there was any evidence of calcification near the lesion. *Healing* [illegible] *healing* [illegible] ing) [illegible] the healing of Pott's disease was concerned with the presence of and the care of the abscess Meyerding agreed that surgical fusion was only an aid and not a cure for tuberculosis of the spine, and believed that fusion could be done without aggravating the disease. *Rest*, a *high caloric* diet and *heliotherapy* are the paramount requirements for these patients [60] A symposium on the subject of Pott's disease which appeared in *The Journal of Bone and Joint Surgery* in 1940 should be of special interest to workers in this field.[2, 3, 6, 14, 17, 36, 42, 50, 58, 83]

MISCELLANEOUS JOINTS

Involvement of the *shoulder or ankle joint*[19, 88] presents no unusual symptoms In the shoulder, arthrodesis is the treatment of choice in adults, and immobilization in abduction in children for months or years.

Tuberculosis of the shoulder must be differentiated from other chronic [illegible] joint [illegible] es, [illegible] ry tuberculosis or local abscess is present. In uncomplicated cases, the operative procedure of choice is extra-articular fusion by an osteoperiosteal graft from the acromion to the neck of the humerus. If postoperative splinting is proper and of sufficient duration, bony ankylosis and intra-articular healing almost always occur in four to eight months. The arm is held slightly forward and abducted to 70°.

Rest and [illegible] also advised in involvement of the elbow joint in child [illegible] early bony c [illegible]

Destruction of a finger joint without new bone formation in the region should be suspected as being of tuberculous etiology.

Peritrochanteric bursae may be involved alone by tuberculosis [47]

destruction while clinically there is little heat or pain in the joint [47] The lesion may heal completely if there has not been complete destruction of joint surfaces.

Juxta-articular Nodes.—These have been reported in patients with syphilitic infection; they may resemble the subcutaneous nodules of rheumatic fever or rheumatoid arthritis.[21]

DIAGNOSIS OF SYPHILITIC JOINT DISEASE

This diagnosis is made by a *careful history* (including a personal or family history of syphilis, repeated miscarriages, interstitial keratitis, iritis, lymphadenopathy, chancre, etc.), a *complete physical examination, blood and spinal fluid Wassermann* and the *clinical manifestations.* The Wassermann test may be negative in known syphilitic bone and joint lesions; on the other hand, *the finding of a positive Wassermann reaction does not necessarily incriminate syphilis as the cause of the joint disease.* Too much credence must not be placed upon a single negative Wassermann reaction if bone or joint syphilis is suspected; in all of these cases, one should *x*-ray the tibia and fibula for additional information.

No pathognomonic roentgen signs of syphilitic arthritis have been reported; suggestive features include erosion of bone and cartilage with bony proliferation and periostitis in a relatively symptomless joint.

Clinical details favoring the diagnosis of syphilitic joint disease include absence of pain, even with marked hydrops, free range of motion, good general health despite a long story of joint pathology, precipitation of the arthritis by a blow (occurring often in syphilitics), other evidence of syphilis, lack of response to salicylates and favorable response to antisyphilitic therapy. *When there is doubt concerning the diagnosis of syphilitic joint disease a biopsy should be performed* [19]

Use of Penicillin in Syphilis.—Penicillin is the treatment of choice in early and late syphilis. Moore, *et al*,[38] and Mahoney and his associates[32] showed that **penicillin had a profound immediate effect in early syphilis** with disappearance of surface organisms from open lesions, healing of lesions and a trend toward serologic reversal. *In primary and secondary syphilis,* one should administer 600,000 units per day for 10 days (a total of 6 million units); This may be given in one daily dose of ordinary penicillin G in oil or crysticillin in saline. There is considerable evidence to indicate that a simultaneously administered course of mapharsen may give fewer relapses [10a]

A favorable effect has also found from penicillin in early asymptomatic [illegible] *t resistant*

[illegible] mmatous

syphilis of the skin and bones healed with a daily dose of approximately [illegible] types of syphilis, penicillin [illegible] the blood in from 50 to [illegible] ent occurred in a high percentage of cases of simple demented paresis and in about 20 per cent of those with tabes dorsalis, *Charcot joint was unaffected by penicillin,* a new one developing during therapy.

27 ERLACHER, P. J.: Jour Bone and Joint Surg, 17, 536, 1935
28 FELDMAN, W F, HINSHAW, H. C, and MOSES, H E Am Jour. Med Sci, 207, 290, 1944
29 FINKELSTEIN, H. *et al.* Jour. Am Med. Assn, 110, 480, 1938
30 FORESTIER, J · Jour. Lab and Clin Med, 20, 827, 1935
31 FRASER, J Tuberculosis of the Bones and Joints in Children, London, A and C, Black, 1914.
32 ———: Jour Exp Med, 17, 362, 1913
33 FRIPP, A. T.: Jour Roy. Inst. Pub Health and Hyg, 3, 127, 1940
34. FRUGONI, C. Ztschr. f Rheumaforsch, 4, 203, 1941
35 GHORMLEY, R K, and DEACON, A. E: Am Jour. Roentgenol, 35, 740, 1936
36 GILL, A B · Jour Bone and Joint Surg, 22, 796, 1940
37 GIRDLESTONE, G R.: Tuberculosis of Bone and Joint, New York, Oxford Univ Press, 1940
38 [illegible]
39 [illegible]
40 [illegible]
41. [illegible]
42 [illegible]
43 [illegible] Jour Bone and Joint Surg, 24, 332, 1942
44 HENCH, P. S, *et al*: Ann Int. Med, 11, 1089, 1938
45 ——— [illegible]
46 ——— [illegible]
47 ——— [illegible]
48 HEND[illegible] Surg, 6, 27, 1936
49 HOUK [illegible]
50 KEY, [illegible]
51 ——— [illegible] 1940
52 KIENBOCK, R · Roentgendiagnostik der Knochen und Gelenkrankheiten, Vienna, 1938
53 KLEINBERG, S. Med. Clin North America, 21, 1723, 1937
54 KÖNIG, F: Die Tuberculose der Menschlichen, Gelenke, sowie der Brustwand unddes Schadel (Aug. Huschaby), Berlin, 1906
55 KUBIRSCHKY: Quoted by Hench, *et al*
56 LIEBOLT, F L Arch Surg, 35, 1005, 1937
57 MAILLAN [illegible]
58. MAYER, I [illegible]
59 MCKEEVI [illegible]
60 MEYERDI [illegible]
61 MITCHELI [illegible]
62 MOLLARD [illegible]
63 MURPHY, [illegible]
64 MURRAY, [illegible] 1940
65 NATHANSON, L, and COHEN, W · Radiology, 36, 550, 1941
66 NICHOLS, E H: Trans Am Orthoped Assn, 11, 353, 1898
67 OBER, F R.· Jour, Am Med Assn, 113, 1299, 1939
68 ORY, M Acta rheum, 4, 8, 1932
69 PAISSEAU, G, and OUMANSKY, V Ann de méd, 26, 30, 1929
70. PEMBERTON, R, and PEIRCE, E. G: Am Jour Med Sci, 173, 31 1927
71 PETTER, C. K Jour Lancet, 15, 117, 1935.
72 PHEMISTER, D B Am Jour Roent and Rad Ther, 12, 1, 1924
73 PHEMISTER, D B, and HATCHER, C H · Am Jour Roent and Rad Ther, 29, 736, 1933
74 PONCET, A · Lyon méd, 99, 65, 1902
75 ———: Bull et mém Soc méd d hôp de Paris, 20, 841, 1903
76 ———· Lyon méd, 111, 237, 1908
77 RAY, N.· Proc Roy Soc Med (Med Sections), 7, 68, 1914
78 ROSENCRANTZ, E, PISCITELLI, A, and BOST, F. C. Jour Bone and Joint Surg, 23, 628, 1941
79 SEDDON, H J Post-Grad Med Jour, 13, 387, 1937
80 SLOCUMB, C H, and GHORMLEY, R. K. Surg Clin North America, 15, 1251, 1935
81 SKOLNICK, M. H · Am Rev. Tuberc, 26, 429, 1937.

Charcot joints may develop gradually or with great suddenness. Although the larger joints are more commonly involved, the small joints of the hands or feet are occasionally destroyed[24] The process is frequently bilateral The disease process may remain stationary for years or may gradually



FIG 200 —Pathologic fracture with ossification in a large hematoma at the left shoulder in a man, aged fifty-two years, with tabes dorsalis The lesion caused very little pain

or tenderness must always make one consider neuroarthropathy.[1]

Roentgen-ray examination of a Charcot joint reveals an enormous destruction of the articular surfaces and of the bones with a chaotic bony and cartilaginous hyperplasia in the surrounding tissues—out of all proportion to the clinical findings[41]

The positive clinical evidence of rigid pupils and absent knee jerks is of much more importance than blood or spinal fluid Wassermann tests in the diagnosis of tabetic arthropathy. The relaxed, disorganized, swollen, comparatively painless joint should always suggest neuroarthropathy; the

an entity such as tuberculous rheumatism. They have shown that: (1) the family incidence of tuberculosis is no higher in polyarthritis than in other non-tuberculous diseases; (2) polyarthritis is very uncommon (from 0 2 to 5 6 per cent) in tuberculous patients;[12,24,77] (3) active pulmonary or other visceral tuberculosis is very uncommon in arthritic patients;[20,40,70] (4) the [illegible]

lesions have been found in the joint tissue on pathologic examination,[8,67] (7) it is difficult to reproduce tuberculous lesions of joints experimentally;[32,66] (8) laboratory tests do not provide adequate proof of the tuberculous nature of the lesion, (9) most workers have been unable to recover tubercle bacilli from culture of these joints or from the blood stream.

It has been pointed out that *it is impossible to distinguish clinically* (including roentgen and physical examinations) *between tuberculous rheumatism and other forms of joint involvement* such as rheumatic fever or rheumatoid arthritis.

Criteria for the diagnosis of tuberculous rheumatism, as enumerated by some workers include:[46,52,55] absence of endocarditis, no response to *salicylates, presence of non-articular tuberculosis, positive tuberculin test,* positive results by animal inoculation from synovial fluid and predominance of mononuclear cells in the synovial fluid.

BIBLIOGRAPHY

1 Adams, R, Jones, G, and Marble, H. C: New England Jour. Med, 223, 706, 1940
2 Adams, Z B Jour Bone and Joint Surg, 22, 860, 1940.
3 ——— Jour Bone and Joint Surg, 18, 974, 1936
4 Adams, Z B, and Decker, J. J · Jour Bone and Joint Surg, 19, 719, 1937
5 Allison, N, and Ghormley, R K · Diagnosis in Joint Disease, New York, William Wood & Co, 1931
6 Amberson, J B, Jr. Jour. Bone and Joint Surg, 22, 807, 1940
7. Anderson, R L · Jour Bone and Joint Surg, 22, 741, 1940
8 Arloing and Lommel. Quoted by Ory.
9 Ashhurst, A P C Surgery, Its Principles and Practice, Philadelphia, Lea & Febiger, 1927
10 Badgley, C E, and Hammond, G · Jour. Bone and Joint Surg, 24, 134, 1942
11. Bray, E A, and Hench, P S Jour Bone and Joint Surg, 16, 839, 1934
12 Brown, L Quoted by Pemberton, R, and Pierce, E G, Am Jour Med Sci, 173, 31, 1927
13 [illegible]
14 [illegible]
15 [illegible]
16. [illegible]
17. [illegible]
18 [illegible]
19 [illegible] New
20 [illegible]
21 [illegible]
22. [illegible]
23 [illegible] de
24 [illegible] Lyon, 13, 265, 1932
25. Duncan, W.· Cleveland Clin Quart, 2, 33, 1935
26. Duncan, W, and Mariette. Quoted by Hench

In the *hips*, there is early erosion of the acetabulum with irregularity of the joint line. Later, one sees enlargement and destruction of the acetabulum and absorption of the neck and head of the femur. In the *knee*, roentgenograms [illegible]
tening [illegible]
varum [illegible]
found in the joint in about half of the cases. In more advanced cases, exostoses are common, in addition to ligamentous and intramuscular ossifications.[43]



FIG 259 —*Charcot joint of a knee* (both knees were similar); patient is sixty years of age. There is marked destruction of the knee joint with marked proliferation and bony fragmentation as is typical of a neurotrophic joint.

The *pathologic process* in general is one of decalcification of bone with gradual destruction of the joint surfaces, followed by an overgrowth of bone at the margins of the joint.[39] It has been mistaken at times for degenerative joint disease[17] The primary pathologic change is an invasion of the articular cartilage by fibrous connective tissue from pannus on its surface. Some effusion is usually present in the joint at the onset; marked edema and infiltration of periarticular tissues may occur. The cartilage breaks up, with formation of multiple loose bodies, and the bone ends literally become ground-down and greatly distorted.

CHAPTER 46

SYPHILITIC JOINT DISEASE AND NEUROGENIC ARTHROPATHY

By Victor G. Balboni, M.D.

True syphilitic arthritis *is not common*. With the use of penicillin in the treatment of syphilis, joint disease has become even more rare It should never be assumed that the mere finding of a positive serology in arthritis is diagnostic of syphilitic joint disease.[19] Joint involvement, however, may occur at any stage of congenital or acquired syphilis[2] Syphilitic joint disease may be classified as follows:

1. **In congenital syphilis:**
   A. *Parrot's syphilitic osteochondritis*

   B. Synovitis.
   C. Hydrarthrosis.
   D. Tenosynovitis.
   E. Bursitis.

' and G may also

soft tissues, cartilage or bone).
   B. Charcot joint (of tabes dorsalis).
   C. Chronic syphilitic arthritis.
   D. Juxta-articular gummata.
   E. Juxta-articular nodes.

In a large series of patients with chronic forms of arthritis, Kuhns and

About 5 per cent of syphilitic children are born dead and only 25 per cent of these congenitally affected survive the first year.

All bone

evidence of

children is

joints, with limitation of motion but very little or no pain on manipulation

Parrot's Syphilitic Osteochondritis (Parrot's Pseudoparalysis).—*The* commonest type of syphilitic joint lesion with objective findings is seen

38 PHILIPS, H B : Am Jour. Surg , 39, 31, 1925.
39 POTTS, W J Ann Surg , 86, 596, 1927.
40 SOTO-HALL, R , and HALDEMAN, K O.: Jour Am. Med Assn , 114, 2076, 1940
41 SUTHERLAND, C G Radiology, 4, 355, 1925
42 SMITH, C B West Virginia Med. Jour , 34, 111, 1938
43 STEINDLER, A : Urol and Cutan. Rev , 49, 568, 1945
44. STOKES, J. H · Modern Clinical Syphilography, 3d ed , Philadelphia, W B Saunders Company, 1944.
45 STOKES, J H , *et al* Jour Am Med Assn , 126, 73, 1944
46 TODD, A H.· Brit Jour Surg , 14, 260, 1926
47 ULLMO, A · Urol and Cutan. Rev , 38, 716, 1934
48 WILE, U. J , and BUTLER, M G · Jour Am Med Assn , 94, 1053, 1930
49 WOLDENBERG, S C · Urol and Cutan Rev , 45, 252, 1941

82. SWEET, P P, BENNETT, G E, and STREET, D. M.: Jour. Bone and Joint Surg, **22**, 878, 1940.
83. SWIFT, W E · Jour Bone and Joint Surg, **22**, 815, 1940
84 TAN, Y. D: Chinese Med Jour. **49**, 293, 1935
85 TORBETT, J W, JR Texas State Jour Med, **31**, 200, 1935
86. TREGUBOV, S J. Jour. Bone and Joint Surg, **35**, 734, 1937
87. VAN BREEMEN, J Acta rheumatol., **4**, 17, 1932.
88 WASSERSUG, J D.. New England Jour. Med, **227**, 436, 1942.
89 BICKEL, W H, YOUNG, H. H, PFUETZE, K H, and NORLEY, T.· Jour Am Med Assn., **137**, 682, 1948
90. BOSWORTH, D M, *et al* Quart. Bull. Sea View Hosp., **13**, 52, 1952
91. Editorial Jour Am. Med Assn, **148**, 1034, 1952.
92. GIRDLESTONE, G R, and SOMERVILLE, E W.: Tuberculosis of Bone and Joint, Second Edition, New York, Oxford Univ. Press, 1952
93 KING, D S · Med Progress—Tuberculosis. New England Jour. Med, **243**, 530, 1950
94. Editorial. New England Jour Med, **246**, 468, 1952

absence of pain (in most instances) despite such an advanced pathologic process is probably the most striking clinical feature. In tabetic cases, one may also note the Argyll Robertson pupils, the positive Romberg, absence of knee jerks and decreased sensation in the lower extremities

The blood Wassermann has been negative in as many as 55 per cent of cases in some series of tabetic arthropathies, while the spinal fluid Wassermann was negative in one-third of these cases.

Specific therapy has no effect upon the articular picture. Charcot joints are treated by rest and immobilization with splints or braces, physical therapy and stabilization and alignment of the joint by conservative or surgical measures Fusion operations are successful at times if the destruc- [illegible] cessary occasionally [illegible] in the stump is not [illegible]

BIBLIOGRAPHY

1 [illegible]
2. [illegible]
3 [illegible]
4 [illegible] 6, 122, 1925.
5 Charcot, J M · Arch de physiol. norm et path, 1, 161, 1868
6 Chesney, A. M, Kemp, J. E, and Resnik, M.· Bull. Johns Hopkins Hosp, 35, 235, 1924
7 Chesney, A M, Kemp, J. E, and Baetger, F. H.· Jour Clin. Invest, 3, 131, 1926.
8 Clutton, H. H Lancet, 1, 391, 1886
9 [illegible] 940
[illegible]
17 Gaston, P. Paris méd, 47, 200, 1923
18 Hatch, E. S · South Med Jour, 24, 471, 1931
19 Hench, P S, *et al* Ann Int Med, 28, 66, 1948
20 Hodges, P C, Phemister, D B, and Brunschwig, A: The Roentgen Diagnosis of Diseases of Bones and Joints, New York, Thomas Nelson & Son, 1938
21. Jeanselme, E Paris méd, 51, 242, 1924
22 Jostes, F A, and Roche, M B Jour Missouri Med Assn, 36, 61, 1939
23 Kernwein, G, and Lyon, W. F · Ann Surg, 115, 267, 1942.
24 Key, J A Am Jour Syph., 16, 429, 1932
25 Klafton, E, and Priesel, R. Fortschr. a d. Geb. d. Rontgstr, 47, 59, 1933.
26 Klauder, J V., and Robertson, H F · Jour Am. Med. Assn, 103, 236, 1934
27. Kuhns, J G, and Feldman, T Urol and Cutan Rev, 44, 532, 1940.
28 Kling, D H Am Jour Med Sci, 183, 538, 1932.
29 Lloyd-Williams, I H Urol and Cutan Rev, 35, 225, 1931.
30 Mahoney, J F, *et al* Jour. Am. Med Assn, 126, 63, 1944
31 McCord, J R. Am Jour Obst and Gynec, 42, 667, 1941.
32 [illegible] 22, 1275, 1938
[illegible] 02, 53, 1934
[illegible] 27, 435, 1938

38 PHILIPS, H B: Am Jour Surg, 39, 31, 1925
39 [illegible], W J: Ann Surg, 86, 506, 1927
d Assn, 114, 2076, 1940
ed, Philadelphia, W. B. Saunders Company, 1944
45 STOKES, J H, *et al*: Jour. Am. Med Assn., 126, 73, 1944
46 TODD, A H: Brit Jour Surg, 14, 260, 1926
47 ULLMO, A: Urol. and Cutan Rev, 38, 716, 1934
48 WILE, U. J, and BUTLER, M G Jour Am Med Assn, 94, 1053, 1930
49 WOLDENBERG, S C Urol and Cutan Rev, 45, 252, 1941

almost 14 per cent of patients ill with diseases other than brucellosis and in more than 10 per cent of apparently healthy subjects[57] These may be false ... repeated contacts with Brucella ... drinking infected milk with the ... persensitiveness (with a positive brucella skin reaction) although the patient had never noticed symptoms of the disease. The allergic state may persist for some years following complete recovery from the disease

Darley found that 15 per cent of "ailing" and 9 per cent of ...

... positive in more than 80 per cent of patients ill with chronic diseases other than brucellosis[57] ... sistent correlation betw... state of infection. In ... organism, may occur even though the agglutination and skin tests as well as the phagocytic index are negative Unfortunately, recovery of Brucella by culture or animal inoculation can be accomplished in only a few cases with brucellosis.

The average duration of undulant fever is *three months, individual cases may persist for years.* There is no pathognomonic type of fever,[51] chronic cases may be almost afebrile.

... *per cent* ... al thou-...

... involvement.[... 165] Arthralgia, non-suppurative or suppurative arthritis intermittent ... sp... ar... Sp... swellings of the fingers may resemble rheumatoid arthritis[163] and migratory joint pains may be mistaken for rheumatic fever ... Bu...

th... Neuralgic pains and transient arthr... arthritis are not ...

... serous or purulent joint ... although the shoulders, knees, hips, ankles and joints of the hands and feet have been affected most commonly Severe pain may be noted in the sacro-iliac joint.[42] A burning type of pain may occur in the wrists, ankles ...

Phalen, Prickman and Krusen reported 3 cases of Brucella spondylitis in two of which destructive lesions of the spinal column were present.[165]

CHAPTER 47

OTHER FORMS OF INFECTIOUS ARTHRITIS

By Victor G. Balboni, M.D.

ACUTE SUPPURATIVE ARTHRITIS

Joint disease from pneumococci, meningococci and hemolytic streptococci is becoming very uncommon since the introduction of modern antibiotics.[99]

Acute suppurative arthritis may be caused by several mechanisms: (1) direct wounds of the joints; (2) blood borne infection (metastatic); and (3) extension from neighboring areas of infection (as from an adjacent steomy ... most nt ... ij eli- ... arlet ,[27] measles, infected wounds, peritonsillar abscess, otitis media,[87] stoid infection, infected varicose ulcers,[173] furuncles, infected burns,[238] pyelitis, infection of the umbilicus and sinusitis.[190]

Bacteria which may produce a purulent arthritis[92,140] include the staphylococci (aureus and albus), hemolytic or green producing streptococci,[221] gonococci, pneumococci, and less common invaders such as the meningococci, influenza bacilli,[184] typhoid bacilli, Bacillus pyocyaneus, micrococcus tetragenus,[191] Salmonella suipestifer,[242] etc.

The pathologic changes vary in intensity with the type of organism and the resistance of the individual,[69 71 131] there may be a mild synovitis or a markedly destructive lesion of the joint which may prove fatal The damage is produced in large part by the proteolytic enzymes of the white blood cells (in the pus), and to a lesser degree by mechanical pressure and erosion.

... include the acute onset of fever, chills, sweats, leuko- ... notion (due to muscular ... The most commo affected areas are the large joints such as the hip,[7,31,65] ... ho nd less commonly an ankle, elbow, sacro-iliac regio ... [92] disease is usually monarticular. Purulent arthritis i children than in adults. Such patients have often bee rheumatic fever or a mild form of infectious arthritis

Nicholson has reviewed the subject of septic arthr to the introduction of the sulfonamides, crippling 87 per cent of these patients. The pyogenic infe borne due to staphylococci or streptococci most of cocci, mixed infections and rarely the pyocy melitensis organisms.

Symptoms include a history of primary infect and often local trauma the preceding day; m

All of the patients recovered and the bony lesions were repaired spontaneously following a course of physically induced hyperpyrexia.

Spondylitis is not uncommon in undulant fever.[21,141,143,179,223,226] Palagi collected 29 cases of brucella spondylitis from the literature in 1934 and added 17 of his own.

As a complication of undulant fever, spondylitis is most likely to occur several months after the beginning of the febrile state but localization in the spinal column has occurred as early as three weeks and as late as a year after the original infection. In some instances, the patient apparently recovered from the systemic disease but was later affected by the spinal complication One should always suspect the possibility of brucellosis with spondylitis in backache occurring in farmers, veterinarians, butchers, dairymen, packing house employees and those with a history of ingestion of raw milk.

Destructive lesions of the vertebrae may occur in brucellosis with wedging of the vertebrae and narrowing of some of the interspaces. *Such lesions are usually limited to a few vertebrae in the lumbar region.*

Steinberg[224] recently reported a case of unilateral sacroiliac arthritis caused by brucella infection. Although previous reports had mentioned

bilateral

In most cases of Brucella spondylitis, the diagnosis is based upon the finding of vertebral lesions or clinical signs and symptoms of spondylitis in

Treatment.—Aureomycin is the drug of choice in all types of brucellosis It is administered orally in doses of 0 5 grams every six hours (2 grams a day). In uncomplicated cases, a fourteen-day course of therapy is often sufficient, but more prolonged therapy or repeated courses may be necessary In cases resistant to aureomycin or those that recur following cessation of treatment, sulfadiazine is given in doses of four to six grams a day in conjunction with aureomycin. Streptomycin is less effective than aureomycin in most cases, but may also be effective in some cases when combined with sulfadiazine. In some cases of brucella spondylitis, surgical intervention may be required, such as incision and drainage of an abscess, or spinal fusion

ARTHRITIS WITH CHRONIC ULCERATIVE COLITIS

Arthritis is an uncommon complication in patients with chronic ulcerative colitis, occurring in about 4 *per cent of cases* [9,32] It is more apt to

an especially important primary focus These patients develop a fever of from 101° to 103° F., moderate leukocytosis and pain, the latter renders

tion, flexion and external rotation. The acute pain permits only a few degrees of motion of the joint. The joint fluid causes bulging and thickening of the capsule; the thigh appears edematous and the inguinal crease is obliterated. Marked tenderness is experienced on local pressure



FIG 261 —Infectious arthritis Note the loss of articular cartilage at points indicated by the arrows and about all of the carpal bones Characteristic area of erosion at *A* General rarefaction of the carpus and articular ends of the long bones (Holmes and Robbins)

Aspiration may decompress the joint, temporarily relieve pain and aid in diagnosis Lateral aspiration is usually employed, the needle is introduced above the tip of the trochanter and is tilted down along the femoral neck. If the infection is permitted to progress, the joint cartilage undergoes autolysis and the distended capsule may rupture at its inferior mesial portion Spontaneous dislocation of the hip may occur (posterior and upward with the head anterior).

accompany subsequent exacerbations of the colitis than the first attack

The joint involvement may be monarticular or polyarticular and varies in severity (usually in proportion to the intensity of the ulcerative colitis)

not been noted.[33]

Some of these patients have had marked improvement in their arthritic phenomena following successful treatment of the chronic ulcerative colitis In other instances, deformities have developed The involved joints are treated as in uncomplicated rheumatoid arthritis.

MISCELLANEOUS FORMS OF INFECTIOUS ARTHRITIS

Infectious Hepatitis.—In 1897, George Still called attention to the fact that during the course of intercurrent hepatitis an improvement of the

In

ronic

with.

hepatitis. A third circumstance often observed in infectious hepatitis is that some cases of hepatitis are introduced by arthritic symptoms lasting from days to months. These symptoms are not characterized only by pains in the joints, but also by swellings, so that clinically they may simulate true rheumatoid arthritis. When the hepatitis of these patients manifests itself by jaundice, the rheumatic symptoms usually disappear In one series, 12 per cent of 485 cases of hepatitis suffered joint symptoms as an early manifestationof their disease [3a]

Actinomycosis.—Forty-seven cases of *actinomycosis of the spine* have been reported,[129,160] this may be mistaken for Pott's disease It is usually secondary to a focus in the respiratory or intestinal tract Clinical diagnosis is very rarely made *Multiple sinuses* may be present and there is a more destructive invasion than in Pott's disease. The mortality rate is about 90 per cent. Dobson and his associates reported cures in 3 cases of actinomycosis by therapy including sulfanilamide, iodides and roentgen-rays. Penicillin is also reported to be of value.[107]

Anthrax.—Joint complications are rare [33,50] Migratory arthralgia and serous exudates have been noted.

Bacillary Dysentery.—Serous effusions containing Shiga's bacillus have been found in approximately 3 per cent of cases of bacillary dysentery [100] This may involve one or multiple joints[156,171,203] and appears most often from one to three weeks after the onset of the diarrhea, although it has *also been noted* several weeks after cessation of the diarrhea [15] *Such* involvement (usually of the knee or ankle) persists from four to six weeks, and is usually followed by complete recovery of joint function.[12,80] Suppuration and resultant ankylosis have occasionally been noted.[95] Kruspe has discussed the possible rôle of dysentery in the etiology of Reiter's disease [139]

Blastomycosis.

CHAPTER 47

OTHER FORMS OF INFECTIOUS ARTHRITIS

By Victor G. Balboni, M.D.

ACUTE SUPPURATIVE ARTHRITIS

Joint disease from pneumococci, meningococci and hemolytic streptococci is becoming very uncommon since the introduction of modern antibiotics.[99]

Acute suppurative arthritis may be caused by several mechanisms: (1) *direct wounds of the joints;* (2) *blood borne infection* (metastatic); and (3) extension from neighboring areas of infection (as from an adjacent osteomy[illegible]
frequent [illegible]
tis,[28 196] [illegible]
fever,[27] [illegible]
mastoid infection, infected varicose ulcers,[173] furuncles, infected burns,[238] pyelitis, infection of the umbilicus and sinusitis.[190]

Bacteria which may produce a purulent arthritis[92,140] include the staphylococci (aureus and albus), hemolytic or green producing streptococci,[221] gonococci, pneumococci, and less common invaders such as the meningococci, influenza bacilli,[184] typhoid bacilli, Bacillus pyocyaneus, micrococcus tetragenus,[191] Salmonella suipestifer,[242] etc.

The pathologic changes vary in intensity with the type of organism and the resistance of the individual;[69 71 131] there may be a mild synovitis or a markedly destructive lesion of the joint which may prove fatal. The damage is produced in large part by the proteolytic enzymes of the white blood cells (in the pus), and to a lesser degree by mechanical pressure and erosion.

Early symptoms include the acute onset of fever, chills, sweats, leukocytosis, *local pain, swelling, redness, limitation of motion (due to muscular* spasm) and tenderness around the joint.[63,96,133 147] The most commonly affected areas are the large joints such as the hip,[7,31,65] knee or shoulder, and less commonly an ankle, elbow, sacro-iliac region or wrist.[102] The disease is usually monarticular. Purulent arthritis is more common in [illegible] as having [illegible]
[illegible] [170] *Prior* [illegible]
[illegible] curred in 87 per cent of these patients The pyogenic infection was usually blood *borne due to staphylococci or streptococci most often,* but also to pneumococci, mixed infections and rarely the pyocyaneus, colon, typhoid, or melitensis organisms.

Symptoms include a history of primary infection of several days' duration and often local trauma the preceding day; mastoid infection in children is

to either sulfanilamide or roentgen therapy[200] The main reservoir for this infection lies in the Sacramento-San Joaquin Valley in the southern agricultural valley of California. About 90 per cent of reported cases have concerned patients observed in California. The disease is caused by the *fungus C. immitis.*

Joint involvement may occur in the acute benign phase of the disease or in the more dangerous chronic granulomatous stage. The disease (also called *valley fever, desert fever, desert rheumatism*) is usually mild and non-fatal and is characterized by an *acute onset with general aches and pains, malaise, fever, sore throat, toxic erythema* and in some instances, conjunctivitis and bronchopneumonia. *Lesions of the erythema nodosum type appear,* especially on the shins from eight to fifteen days after the onset of the disease. Pulmonary lesions may appear at this time.

Joint involvement is *relatively insignificant,* although it may be distressingly painful; *the arthritis subsides completely.* If the joints are involved in the granulomatous phase of the disease, this is serious and may be an indication of a disseminated infection which will lead to death.

About a third of the patients with valley fever develop signs of acute arthritis. This usually appears simultaneously with the lesions of erythema nodosum. The *affected joints are tender, painful on motion and may be slightly swollen.* No one has observed effusion into these joints nor has suppuration taken place. In some instances, *arthritis, conjunctivitis and erythema nodosum* appear together, persist approximately a month and then disappear at about the same time. There is no residual damage or deformity in the affected joints.

One should suspect the presence of valley fever especially in those with acute arthritis which is accompanied by an acute pulmonary infection with or without the presence of erythema nodosum. If this patient has [illegible] the disease is much more [illegible] ving the C immitis [illegible] *ion into guinea pigs*

In patients with the acute type of valley fever, survival is the rule *The chronic granulomatous phase develops in only about 1 in every 400 patients with valley fever.* In these chronic granulomatous stages (coccidioidal granuloma) the mortality may be as high as 50 per cent.

In this chronic granulomatous phase of coccidioidomycosis, *bone and* [illegible] of 256 cases, the joints were [illegible] 16, [illegible] n 5, [illegible] *in* [illegible] *these joints may mimic those of tuberculous arthritis* with regions of destruction in articular surfaces early which is often accompanied by swelling of the overlying soft tissues Articular cartilage may be destroyed with narrowing of the joint spaces. The joint spaces may completely disappear in later stages and extensive areas of destruction may be noted in articular surfaces. *Ankylosis may occur* in some cases.[158] The arthritis is predominantly destructive both in tuberculosis and in coccidioidal granuloma with little tendency to heal by production of bone; pulmonary involvement is frequent in both. Coccidioidal granuloma usually attacks *joints by extension* from adjacent bony lesions.[35] The destructive process in bones in coccidioidal granuloma develops more rapidly than does tuberculosis.[233]

an especially important primary focus. These patients develop a fever of

tion, flexion and external rotation. The acute pain permits only a few degrees of motion of the joint The joint fluid causes bulging and thickening of the capsule; the thigh appears edematous and the inguinal crease is obliterated Marked tenderness is experienced on local pressure



e e d s d- ry tra- ss in eating joint as

ead can be nces, sary t insid

autolysis and the distended capsule may rupture portion. Spontaneous dislocation of the upward with the head anterior)

contain several hundred thousand leukocytes *per c.mm. in hemolytic* streptococcal arthritis; almost all of these cells may be polymorphonuclear cells [132] This should be considered in any case of acute monarthritis with severe symptoms in which moderate or marked constitutional symptoms are present [162, 176]

Hefke and Turner have called *attention to obscuring or widening and* curving of the border of the obturator shadow as seen by *x*-ray in some types of hip joint pathology; this is called a positive obturator sign.[97] It was thought that this sign reveals the presence of septic hip joint disease by *x*-ray at an early stage, and is of some importance in the early diagnosis of tuberculosis of the hip. *It is absent in fractures about the hip,* osteochondritis and other pathologic conditions of the hip.

The obturator shadow is a soft-tissue shadow seen in the A-P roentgen examination of the pelvis; it is just medial to the acetabulum on the inner aspect of the pelvis. The upper border of the shadow usually begins *lateral to and below the lower pole of the sacro-iliac joint and follows* the ... the acetabulum, disappearing behind ... believed that the substratum of the ... iose tendon is adjacent to the capsule of the hip joint.

Pyogenic arthritis of the sacro-iliac joint is very unusual. Avila reported 7 cases of primary pyogenic infection of this joint with recovery in all. In all of these cases but 1, the abscess formed anteriorly.[5,118,148]

Seven cases of septic spondylitis were reviewed, 5 of which were caused by staphylococci, 1 by streptococci and 1 possibly by the influenza virus Six cases were acute, with abscesses and 1 was chronic, without an abscess The *x*-ray revealed the abscesses and edema early. Treatment consisted of incision of the abscesses and immobilization of the affected part. Healing occurred in every case, but some stiffness of the back persisted

In cases of marked infection, penicillin cannot always control infection alone, but surgical procedures may still be necessary.

... monarticular phenomena, but in ... mild. Acute ... ity arthritis, constitutional reaction is also usually mild. The existence of only a ... rthritis must be ruled out

... is instituted within the

be the main determining factors. The mortality reached ... pre-sulfonamide era.

Treatment.—With multiple antibiotics to choose from, it is important that the most effective one is selected. To do this, one must identify the

fevers.[3] [illegible]
to be the [illegible]
an entire [illegible]
moniliformis, Streptothrix muris ratti) has been *recovered from a few of* these patients.

Haverhillia multiformis is also the organism recovered from patients with Haverhill fever, although there was no history of rat bite in the individuals in the epidemic at Haverhill in 1926.

Sodoku is the name given by the Japanese to infection with Spirillum minus. Joint involvement (arthritis) has been mentioned in only a few *patients with this disease. Fevers due to Haverhillia multiformis* are characterized by septicemia with metastatic arthritis and morbilliform and petechial cutaneous eruptions. Penicillin is effective in the therapy of rat bite fever due to either organism.[107]

Histoplasmosis.—Histoplasmosis is a generalized disease with variable clinical characteristics, it is caused by the histoplasma capsulatum of Darling.[184] The four *main clinical pictures* of histoplasmosis include: (1) *a febrile illness with enlarged liver and spleen, anemia, leukopenia* and septic temperature; (2) predominance of lymphadenopathy, simulating Hodgkin's disease, leukemia or lymphosarcoma; (3) predominance of pulmonary symptoms, often complicated by tuberculosis and (4) onset with a small skin lesion which may develop into a generalized ulcerative cutaneous lesion.

Key and Large reported the occurrence of *involvement of a knee joint by histoplasmosis*, which closely resembled tuberculosis of the knee joint [illegible] disability in the left knee [illegible]

intense pain on movement.

The knee was considerably enlarged, and excess fluid was present in the joint. The periarticular tissues were thickened. No redness was noted but there was a moderate increase in local heat over the joint.

Roentgen examination of the knee showed thickening of the soft tissues and moderate bony atrophy with spotty atrophy of the bone structure.

A thick purulent material was aspirated from the knee joint; culture of this was negative. Inoculation of the material into guinea pigs failed to show tuberculosis.

X-rays taken four weeks later showed increased atrophy of the bone, narrowing of the joint space and marked broadening and erosion of the margins of the intercondylar notch.

The extremity was amputated as the patient did not respond to sulfonamides or other therapy.

On gross examination, the knee was markedly enlarged. The synovial cavity resembled a large abscess cavity, filled with thick, gray pus; the abscess extended into the calf. There was considerable erosion and necrosis of [illegible] resembled advanced *tul*[illegible]
tis[illegible]

no tubercles were seen and the giant cells were of the [illegible]
No acid fast organisms were seen. The synovial membrane was trans-

end of the first year of life when one notices the hip lurch produced by the dislocation.

Harmon has described a reconstruction operation which may be done during the second and third years of life.[89]

Epiphyseal pseudoarthrosis at the hip may be seen after operative fusion of the hip, following inflammatory ankylosis of the hip or as a sequel to adolescent epiphysiolysis [162]

In septic arthritis of the hip joint, rest of the joint should be accomplished by a double hip spica including the thigh of the opposite leg to prevent adduction and abduction, and the foot on the involved side to control *rotation Windows may be provided in the cast for aspiration of the joint.* In some cases, surgical drainage may be necessary (if the femoral head is destroyed, if thick purulent material is present, etc.) Dependent drainage may be obtained by opening the hip joint posteriorly. Weight bearing must not be instituted too early, follow-up orthopedic care and physical therapy are extremely important

Heberling reviewed the results of treatment of 201 patients with acute suppurative arthritis [96] Surgical therapy consisted of incision of the joint,

Early walking and weight bearing did not hasten recovery In those with involvement of the knee or hip, adhesive traction with a small amount of *weight was used.*

Sites of incision were as follows (1) Shoulder: anteriorly through the deltoid. (2) Elbow. posteriorly over the radio-humeral region (3) Wrist: dorsal into the snuff-box. (4) Hip. anteriorly between the sartorius and the tensor fasciae femoris (5) Knee: mesial anterior and occasionally a second incision on the lateral side of the patella (6) Ankle: posterolateral and anteromesial.

After opening the joint, it is gently flexed and extended a few times to

the joint begin to disappear, the drains are removed and the opening is allowed to close gradually.

JOINT INVOLVEMENT IN UNDULANT (MALTA) FEVER (*BRUCELLOSIS*)

Brucellosis frequently exhibits joint pain, pains in the limbs or "rheumatic pains." Arthralgia is common in brucellosis but actual arthritis is rare; in occasional cases, the joint symptoms may resemble those of rheumatic fever or rheumatoid arthritis [99] Hydrarthrosis occurs occasionally

Green and Freyberg studied the incidence of brucellosis in 25 patients with rheumatoid arthritis and in 25 patients with other rheumatic disorders.[77] None of the patients with rheumatoid arthritis had evidence hich was diagnostic of brucellosis. In the second group, 3 had convincing idence of active brucellosis and 6 others had "possible brucellosis"

formed into a mass of granulation tissue, infiltrated with giant cells and macrophages Monocytes or mononuclear phagocytes were the predominating cells in the inflammatory exudate. *Many of the phagocytic cells contained the histoplasma capsulatum of Darling* These parasites were present in enormous numbers in some places.

Almost all recorded cases of histoplasmosis have died (with only several exceptions). Full doses of sulfathiazole had no effect in Key's case. *Meleney has suggested the use of antimony preparations.*

Infectious Mononucleosis.—Although arthralgia is unusual in this disease, joint pain and fever may occasionally be the symptoms at onset and may simulate rheumatic fever.[17]

Influenza.—Monarticular or polyarticular joint involvement may complicate influenza,[23,63,155] this usually appears early in the course of the disease, but may not be noted until several weeks later. Suppuration has occasionally occurred.[184] Although joint involvement has been reported in as many as 25 per cent of such patients,[250] the present concept is that such joint pathology is much less common now [95]

[illegible]

asymptomatic and heals spontaneously. Joint involvement may occasionally resemble rheumatic fever or purulent arthritis [125]

The arthritis which may occur with lymphogranuloma venereum runs a variable course In occasional cases, one may encounter tender, painful, *acutely swollen joints*, however, the process is *usually chronic* and *persists for weeks or months.* Polyarticular involvement is common, with frequent involvement of the knees, ankles and wrists Intermittent hydrarthrosis occurs occasionally The Frei reaction is positive in all cases, cultures of synovial fluid are sterile [99]

Dawson and Boots have reported a series of 24 patients (16 females and 8 males) who showed an arthritis associated with lymphogranuloma venereum.[46] In most of these individuals (usually colored), there occurred a *chronic indolent serous arthritis*, often with intermittent hydrops, acute flare-ups were not uncommon and there was a tendency to relapse The disease was usually polyarticular, involving especially the knees, ankles, shoulders and wrists, occasionally, migratory joint involvement or generalized arthralgia occurred Bilateral joint involvement was noted in 50 per cent of the cases Patients varied from nineteen to fifty-five years of age, the majority being in the third and fourth decades

This type of joint involvement *persisted for weeks or months without destruction of bone or cartilage;* swelling was usually confined to the periarticular tissues The *joint fluid was sterile on culture, and was never purulent* Cell count of the synovial fluid *varied from 900 to 5000 per c mm*

Other findings in these patients included *A positive Frei skin test*, other evidences of lymphogranulomatous infection (especially *rectal strictures*); marked elevation of the sedimentation rate; and a positive gonococcus complement fixation test in 33 per cent of cases.

Grace and Rake have employed a new yolk sac antigen consisting of a suspension in isotonic solutions of sodium chloride of the elementary bodies of the virus of venereal lymphogranuloma; this can be prepared with any desired concentration of virus [78,79] In performing *complement*

necessary. Corrective orthopedic surgical procedures are required in ... early and judicious ... intervention (except ... streptomycin) should become unnecessary in most of these cases.

Treatment of Suppurative Arthritis
1. ... Gram-positive bacteria, the meningococcus and the gonococcus. Streptomycin is used initially in all Gram-negative bacteria except the meningococcus and gonococcus.
2. Penicillin, 1,000,000 units or more daily intramuscularly in divided doses every three hours for a minimum of ten days.
3. Streptomycin, 0.5 grams, intramuscularly every six hours (two grams a day) for a minimum of ten days.
4. Later change to one of the broad-spectrum antibiotics if clinical improvement has not occurred, drug selection being made on basis of in vitro sensitivity tests.
5. Aspirate joint daily with large bore needle, removing all pus until fluid no longer recurs or ceases to be purulent. Instil 20,000 units of penicillin, or 5 milligrams streptomycin after each aspiration if one of these drugs is being used parenterally.
6. ... needle.

Orthopedic Management

Suppuration within the hip joint runs a benign acute course during the first two years of life compared with this disease in later childhood and

fixation tests with this antigen, it was found that these tests were more sensitive than the Frei reaction.

Dawson and Boots believed that the arthritis was a clinical entity associated with *lymphogranuloma venereum; however*, the specific virus was never demonstrated in the joint fluid.

This disease often responds to sulfonamide therapy.[85,183,208,234] Visceral and joint manifestations have usually disappeared following sulfonamides. Despite this, *a positive Frei test may persist for many years after apparent healing* has occurred (as is the case with the tuberculin test in tuberculosis), *in the early stages, the Frei test is negative.*

Aureomycin has been found to improve markedly the acute ulcerative and inguinal forms of this disease.

Measles.—Although articular complications have been reported occasionally, their association with measles has not been definitely accepted. Infectious spondylitis has occurred occasionally after an attack of measles.[4]

Meningococcal Arthritis.—Gwyn in 1898 reported the first authentic case of meningococcus septicemia in a patient with meningitis and arthritis.

Articular lesions have developed in from 5 to 20 per cent of meningococcus infections in various epidemics.[124,202]

Meningococcus infections increase in number in war times. The conditions at large training stations are ideal for the production of a state in which these infections are endemic: men live in barracks and new recruits are constantly arriving. Bush and Bailey discussed an epidemic of meningococcus infection at a Naval Training Station.[30]

Jaeger found 37 cases of meningococcic arthritis among 1000 cases of meningococcic infection.[123] It is very likely that the true incidence of meningococcic joint involvement was much higher inasmuch as when *postmortem examination was made in 40 cases, 9 showed purulent synovitis* which was not diagnosed clinically. Instances of purulent *meningococcic bursitis* and *purulent tenosynovitis* were described. This study suggests that most cases of meningococcus arthritis are instances of *purulent synovitis*. The *wrists* were involved most often; *knee joints* were also frequently attacked. The onset of the disease was either as a *mono-* or a *polyarticular involvement*, the cases being about equally divided in the two groups. The *prognosis was good in all cases.*

During non-epidemic periods, from 2 to 10 per cent of troops may be *meningococcus carriers*[49] while in periods of epidemics, the rate in healthy troops may reach 80 per cent.[37] The organism probably gains entrance to the body from the nasopharynx with subsequent invasion of the blood stream, and if not prevented by spontaneous resistance or therapy, localizes in the meninges, joints, skin, eyes or other body tissues.[109] The idea *has been abandoned* that meningitis is due to direct infection of the meninges through the cribriform plate except in a few cases where known trauma, with fracture, is present.

Meningococcus sepsis usually begins with *prodromal symptoms of disease of the upper respiratory tract.*[222] From one to many days later, *acute symptoms* appear such as a *chill*, with *fever;* the latter may rise suddenly or this may be gradual. *Other symptoms* then include weakness, malaise, *muscular aches*, *headache*, nausea, *vomiting*, and *pains* or actual inflammation *in joints*. *The rash is the most characteristic manifestation;* the most common manifestation of this is *petechial* or *purpuric* with lesions varying

end of the first year of life when one notices the hip lurch produced by the dislocation.

Harmon has described a reconstruction operation which may be done during the second and third years of life.[81]

Epiphyseal pseudoarthrosis at the hip may be seen after operative fusion of the hip, following inflammatory ankylosis of the hip or as a sequel to adolescent epiphysiolysis[162]

In septic arthritis of the hip joint, rest of the joint should be accomplished by a double hip spica including the thigh of the opposite leg to prevent adduction and abduction, and the foot on the involved side to control rotation. Windows may be provided in the cast for aspiration of the joint.

are extremely important

Heberling reviewed the results of treatment of 201 patients with acute suppurative arthritis.[86] Surgical therapy consisted of incision of the joint, under general anesthesia and suturing an appropriate sized soft rubber

with involvement of the knee or hip, adhesive traction with a small amount of weight was used.

Sites of incision were as follows. (1) Shoulder: anteriorly through the deltoid. (2) Elbow: posteriorly over the radio-humeral region (3) Wrist dorsal into the snuff-box (4) Hip anteriorly between the sartorius and the tensor fasciae femoris. (5) Knee. mesial anterior and occasionally a second incision on the lateral side of the patella (6) Ankle posterolateral and anteromesial

After opening the joint, it is gently flexed and extended a few times to express the contents before ...

the joint begin to disappear, the drains are removed and the opening is allowed to close gradually.

JOINT INVOLVEMENT IN UNDULANT (MALTA) FEVER (BRUCELLOSIS)

Brucellosis frequently exhibits joint pain, pains in the limbs or "rheumatic pains." Arthralgia is common in brucellosis but actual arthritis is rare ...

with rheumatoid arthritis and in 25 patients with other rheumatic disorders[82] None of the patients with rheumatoid arthritis had evidence which was diagnostic of brucellosis In the second group, 3 had convincing evidence of active brucellosis and 6 others had "possible brucellosis"

from 1 to 15 mm. in diameter. Many other forms of skin lesions may appear.

The articular complications of cerebrospinal fever were first reported by Welch Osler[174] reviewed this literature and Rolleston[198] and Herrick and Parkhurst added valuable information gained during World War I [111] Schein reported personal experience with 23 cases [210] In World War II, Strong and Hollander[230] reported 100 cases of meningococcic infection In their series, 11 per cent had joint symptoms and in 4 per cent suppurative arthritis developed. *In 1 case the diagnosis of the infection was made from* recovery of the organisms from the joint fluid because blood and spinal fluid cultures were negative Many of the other cases had bacteremia without meningitis

This may occur as pain and tenderness in multiple joints, usually without much effusion [11] Hemorrhages may occur into articular or periarticular tissues (usually causing only transitory involvement)

Keefer feels that meningococcic arthritis is a metastatic lesion [131]

Pyarthrosis due to the meningococcus most commonly appears at the end of the first week of the disease. This usually involves only a single joint Meningococci may be found in the joint fluid Intermittent migratory arthritis may occur in chronic meningococcal septicemia

Articular involvement in meningococcus infections is of several varieties[66] *One may see arthralgias involving multiple joints* during the first few days of the disease as a part of the meningococcemia—occurring simultaneously with the purpuric manifestations Herrick has described intra- and periarticular hemorrhages of the joints in these cases The involvement is usually symmetrical and is often transitory in nature, producing marked pain, redness and tenderness of the joints but a minimal

In others, the arthropathy begins after the fifth day of the disease and is usually *monarticular—involving particularly the knee, if polyarticular* involvement occurs, one joint is usually involved to a much greater extent than the others. Joint effusion may be marked, the amount of pain, redness and tenderness over the joint varies greatly. Meningococci have been found in these joints in about one-third of the cases The *joint fluid varies from a frankly purulent effusion to a serous, mucinous or sero-purulent affair*

With the passing of serum therapy in meningitis, one no longer sees the arthralgias and arthropathies associated with serum sickness in patients with meningococcic infections.[110] However, joint pain may occur following the use of sulfonamides. This disappears following withdrawal of sulfonamides

Most observers question the existence of the so-called postmeningitic

myelitis or herniation of the nucleus pulposus from an injury of the annulus fibrosis by the spinal puncture needle.[66]

Adams has discussed the *arthritis* which occurs *with meningococcemia.*[2] In the non-meningitis case, there is usually a history of respiratory symptoms and joint pains with or without redness and swelling. Large joints,

They concluded that *although arthralgia and other rheumatic symptoms are common in brucellosis* and in some cases non-purulent joint inflammation occurs as a temporary phenomenon, brucellosis is seldom, if ever a cause of chronic non-purulent joint inflammation.

More than 50 per cent of patients with brucellosis may have rheumatic complaints such as myalgia, arthralgia, non-suppurative or suppurative arthritis, spondylitis, osteomyelitis or osteoperiostitis.[105] In some cases (up to 10 per cent) joint symptoms may appear as the chief complaint[220]

Arthralgia was reported in 32 per cent of one series of patients with brucellosis, but evidence of joint inflammation was found in less than 2 per cent.[88] The incidence and prominence of joint pains is greater in California where the bovine strain of Brucella is the predominant type than in Iowa where the porcine strain predominates.[105 232] The bovine strain causes actual joint inflammation only rarely while the porcine strain produces arthritis more often (but not as commonly as the melitensis strain)

Diagnosis of Undulant Fever.—In most instances, chronic brucellosis is diagnosed by the history, clinical findings, specific laboratory tests for brucellosis and exclusion of other diseases The diagnosis may be suggested

ache, depression, secondary anemia, leukopenia with relative lymphocytosis or normal leukocyte count, positive agglutination test,[18 232] positive *intradermal test,*[13 65 90] *cultivation of organisms from blood*[128,251] *or joints,* opsonophagocytic test[64 76 117] and exclusion of other diseases. *Because of the multiplicity of symptoms and paucity of physical signs, the disease is often overlooked or is labeled neurasthenia.*[56] Occasional cases may resemble pulmonary tuberculosis[145 148 243] or encephalitis[156] Brucellosis should be suspected in any long continued, undiagnosed illness, especially if fever is present.[36]

obtained by *culture of joint fluid.*

Laboratory tests for brucellosis are frequently confusing and may be misleading and permit different interpretation The most reliable is the *agglutination test,* but this is not infallible. In one series, a negative agglutination test (titer less than 1 40) was found in 46 per cent of patients with chronic brucellosis.[57] It must also be remembered that the titer may remain high after recovery from the disease

There is no general agreement as to how strong a Brucella abortus agglutination must be before the condition is considered active. *Most clinicians required a positive reaction in a titer of 1:80 or more as evidence*
often negative in those
The *opsonophagocytic*
licated procedure and
much controversy has arisen regarding its interpretation.[44 219]

The *brucellergin skin test* is *less specific* and therefore *less reliable* than the agglutination test. Positive brucella skin tests have been reported in

such as the knees are affected most often. These individuals will usually also have deep muscle aching in the arms and legs, and a macular or petechial eruption. In chronic meningococcemia, one or more joints may be tender. Early treatment with sulfonamides is almost certain to effect a cure except in acute fulminating septicemia and in those with signs of meningitis who are seen late in the course of the disease

Seventy-six consecutive cases of meningococcus meningitis recovered satisfactorily with sulfonamide therapy, as did 50 consecutive cases of meningococcemia. The joint manifestations all responded to sulfonamide therapy.[30]

In one series, 80 patients with meningococcus meningitis and 32 with meningococcemia have been treated with sulfadiazine with one death in the former group and none in the latter.[43]

It has been suggested that the initial dose of sulfonamide be administered intravenously to patients with meningitis even though they appear only moderately ill when first seen.

Hill and Lever[112] reported 2 cases of unilateral arthritis of the knee joint among 68 patients with meningococcic infection seen in an Army Station Hospital in this country. Both resolved completely within two weeks without tapping, sulfadiazine therapy was used routinely.

Strong and Hollander[230] treated 4 cases of suppurative meningococcal arthritis by [illegible]

These pat[illegible] joints, repeated as[illegible] and/or penicillin should be given [illegible] The prognosis is good when these drugs are used. *After the acute stage of the disease has passed, adequate physical* [illegible] will be of great value and should be [illegible]

[illegible] ur occasionally. Joint pains are not uncommon and usually disappear within a week. Acute arthritis has also followed suppurative parotitis.

Puerperal Septicemia.—Joint involvement is usually the result of metastatic infection via the blood stream, most commonly from hemolytic streptococci. Very mild or severe joint damage may ensue. If due to hemolytic streptococci, good results may be obtained with sulfonamides or penicillin

Scarlet Fever.—Healey reported that 3 per cent of young adults being immunized against scarlet fever developed multiple joint pains as a reaction to one or more injections of the sterile filtrate of broth cultures of scarlet fever streptococci [95] The majority of individuals with this reaction had noted joint pains previously associated with rheumatic fever or streptococcic infections of the nose and throat. Such individuals appeared to develop rheumatic affections such as heart disease, polyarthritis and erythema nodosum more often than other persons not similarly sensitized.[194]

From 1 to 5 per cent of patients with scarlet fever develop joint complications,[199] beginning about the fourth to the tenth day of the disease. These may include a mild, migratory polyarthritis resembling rheumatic fever;[182] purulent arthritis with hemolytic streptococci cultured from the joint fluid; or recrudescence of a rheumatic fever.

Smallpox.—Arthritis resulting from smallpox or vaccination has been observed.[115] Suppurative arthritis and even osteomyelitis apparently was caused by secondary infection of the pustules. Lesions caused by the

smallpox virus in the ends of the bones were described as necrotic areas which led to arrest of bone growth and rarely to ankylosis of a joint

Spirochetemia.—Williams reported the finding of *fusiform bacilli and spirochetes* (of the type found in Vincent's angina) *in the cultures of blood from 2 patients*, in one of these patients, the organisms were recovered from the blood eleven times, and successful subcultures of the organism were made [245] These organisms were slightly pathogenic for rats In these 2 patients with *fusospirochetemia, myalgia and a migratory arthritis was observed*, together with the sudden development of fever, headache and prostration In one of these splenomegaly appeared, with jaundice, pleurisy and a hemorrhagic eruption. The source of the infection was considered to be the bite of a rat in one instance and the tonsils in another

Causative organisms have been cultured from the blood in but a few of the spirochetal diseases [148] Reiter cultured spirochetes from the blood of a patient with cystitis, arthritis and conjunctivitis [193] Palmer demonstrated many times the presence of spirochetes in the blood of a patient [illegible] ing [illegible] ute fever, relapsing fever and Weil's disease Fever developed suddenly associated with chilliness, prostration, headache and muscular and joint aching, followed subsequently by a migratory arthritis The first of these patients had an intermittent type of arthritis for two years, while the second was free of arthritic symptoms after a month In both of these patients *clinical improvement followed treatment with arsphenamine* and fusiform bacilli and spirochetes apparently disappeared from the blood Penicillin is now the treatment of choice [107]

Subacute Bacterial Endocarditis.—A non-suppurative arthritis may occur [illegible] The inflammation [illegible] urulent arthritis due

Typhoid Fever[158 205 203] **and Paratyphoid Fever.**[105 195]—Typhoidal arthritis is uncommon. Monarticular or polyarticular arthritis may occur in as many as 12 [illegible]

[illegible]

The lower portion of the spine is most often affected Chloromycetin is the antibiotic of choice for treatment of these infections

Miscellaneous.—Davidson reported a case of "infectious" polyarthritis associated with chronic bilateral bronchiectasis [45]

Arthritis has also been caused by micrococcus tetragenous infection,[191] B suipestifer[83 224 236] (during the first year of life in a prematurely born infant) [illegible] arthrosis), micr[illegible] —a purulent [illegible] ularemia [61] On[illegible] fluid

[illegible] on of twenty-four years' duration.[52] This organism had been recovered from a patient with purulent arthritis

The soldiers nicknamed their disease "Fox-hole arthritis" or "Bougainville rheumatism." More recently Hench has suggested the more descriptive name "Epidemic Tropical Acute Polyarthritis."[39]

The clinical features of all cases were similar except for minor variations.[34,35,39,40] The onset was gradual, and initial symptoms were pains and sometimes swelling with redness of many peripheral joints. Involvement of midphalangeal joints of the fingers resembling rheumatoid arthritis occurred. A maculo-papular rash somewhat resembling rubella usually followed and occasionally preceded the acute arthritis. Tender enlargement of lymph nodes and a mild fever (99° to 101° F.) were present. The disease was self-limited, rarely lasting more than ten to twenty days, and cleared without articular or cardiac sequelæ.

The etiology is unknown. Agglutination tests for Proteus OX19, XK, and OX2 and for Brucella abortus were negative. Cultures of blood, urine, [illegible]

was found.

In differential diagnosis, there were considered rheumatic fever, Haverhill fever and dengue. Streptoccic infection did not precede the disease, and response to salicylates was poor. The rash was more extensive than that seen in dengue, pains were articular and not osseous and post-dengue exhaustion was absent.

Treatment was symptomatic and supportive.[39]

SPIROCHETAL DISEASES

Bejel, the native, endemic, non-venereal treponematosis of the Euphrates Valley, *frequently exhibits bone changes.* There is periosteal or endosteal proliferation with or without areas of rarefaction.[41,36,43] Gumma-like [illegible]

initial *incubation* period, along with slight fever, there may be headache, pain in the loins, rheumatic-like pains, worse at night, in the joints and long bones. In the *secondary* stage, six to twelve weeks after the primary lesion has appeared, there may again be irregular fever with headache and pains in the muscles, bones and joints. *In late yaws, bones and joints are affected in a fashion reminiscent of syphilis.* With diffuse osteitis and periostitis there are severe bone pains not always distinguishable from pain in the joints.

The *joints may be attacked* with *hydrarthrosis* in some, and in others, chronic infiltration of perisynovial or synovial tissues, tenosynovitis or chondrosynovitis or perhaps disorganization of the whole joint. *Ankylosis may result in a useless finger, hand, large joint or extremity.*

Pharmaceutic Arthritis.—Bone and joint pains, and occasionally even joint inflammation may occur following the administration of various drugs used to treat infections, such as arsenic, mapharsen, iodobismitol, gold, sulfonamides, or penicillin.

Articular Reactions to Sulfonamides.—Long observed the occurrence of painful joints during sulfanilamide or sulfathiazole therapy.[181] Glicklich and Sherman reported a patient with chancroidal infection who developed tender swollen elbows and knees during sulfathiazole therapy. The fluid was sterile and contained only a few polys per high power field. Other manifestations of *drug toxicity* were also present.

The joint pathology subsided with cessation of sulfonamide therapy, and recurred again a week later after administration of a test dose of 3 grams of sulfathiazole; the joint swelling again promptly disappeared within twenty-four hours after stopping the sulfonamide. We have observed this same phenomenon in several patients with meningococcic meningitis who received sulfadiazine, the swollen joints (elbow and knee) contained a sterile fluid and the joint swelling disappeared promptly after withdrawal of sulfonamides, leaving no detectable joint pathology.

Articular Reactions to Penicillin.—These are uncommon, and similar to those seen occasionally associated with sulfonamide therapy. *Painful* polyarthritis, fever, urticaria, or sometimes erythema, adenopathy, and dyspnea may occur. Such reactions usually begin seven to fourteen days after penicillin therapy has been instituted and rarely as soon as forty-eight hours. The joint symptoms subside within four or five days of stopping penicillin. Rarely, transient joint symptoms have started four or five days after cessation of penicillin. The joint symptoms *per se* are not an indica-

BIBLIOGRAPHY

1. Abbott, K B, Abbott, F F, and Abbott, C N: Calif. and West. Med, 47, 408, 1937
2 Adams, F D: Ann. Int Med, 20, 33, 1944
3 Allbritten, F F., Sheely, R F, and Jeffers, W A.: Jour. Am Med Assn, 114, 2360, 1940
"Rheumatic Diseases," W. B Saunders Co,
Dis, 4, 440, 1907.
22, 1941
nd Snyder, C H: Jour Bone
8 Balboni, V G, Shapiro, I, and Kydd, D M: Am. Jour Med Sci, 210, 588, 1945
9. Bargen, J A. The Management of Colitis, New York, National Med Book Company, 1935
10. Bauer, W, and Engleman, E P: Trans Assn Am Phys, 57, 307, 1942
11. Bauer, W., and Short, C L: New England Jour Med, 223, 286, 1940
12 Bazán, F., and Maggi, R. Archiv argent. de pediat, 12, 376, 1939
13. Beatty, O A: Kentucky Med Jour, 35, 260, 1937
14 Beeson, P. B: Jour Am Med Assn, 123, 332, 1943
15 Benjafield, J D., and Halley, G. S: Lancet, 2, 616, 1938

Symptoms are often far fewer than one would expect from the extent of the lesions. A less direct joint involvement is seen in the *bow contraction of the li*
yaws
capsule.
ankle or leg. They may soften and lead to lesions of the bone and deformities with deep ulceration and scarring, especially where the bone is close to the skin. Starting from this, joints may be affected at the shoulder, elbow, wrist, ankle or feet *Juxta-articular nodes* may appear placed symmetrically in relation to joints, as over the olecranon or the lower end of the femur. They are movable at first, later becoming fixed. *Bone and joint sequelæ are important in yaws and in some regions occur in* 20 *per cent of the patients.* Severe pains may be an outstanding feature of the disease.

Relapsing Fever.—The *onset* is usually with pains so severe as to constitute the outstanding symptom. They may be very persistent throughout the course of the fever and recur when the fever recurs; they may persist for a time after the fever subsides There are headaches, *often frontal*, general body aches, gnawing pains at the nape of the neck, pains in the back and loins and in the muscles, bones and joints of the limbs.[14,42 50,53,54 66,79,81,82]

cate convalescence.

Infectious Jaundice or Weil's Disease.—The onset is usually sudden with headache, pain and tenderness in the back and limbs, sometimes agonizing in severity, especially in the calves. There may be joint *as well as muscle pains.* Severe *muscle pain and tenderness are so characteristic as to suggest the diagnosis.*[14 16,53,54 66,82]

Other spirochetal diseases associated with pains in the muscles are seven-day fever where the onset is abrupt, accompanied by pain, and the pseudodengue of Java which gives severe pain especially in the legs [82]

PROTOZOAL DISEASES

Trypanosomiasis.—In the early stages of African sleeping sickness there are headache, neuralgic pains especially in the joints, and cramps [19 54,66,82] *Kerandel's symptom* may be present, this is a deep hyperesthesia with
bony prominence
r joints, some-
ewhere. In the
and a shuffling
gait with weakness of the legs.

Leishmaniasis.—True arthritis is not part of the picture in any of the leishmania infections [8 54]

Malaria.—With the high fever of a malarial attack the patient com-
headache and *pains in the back and in the joints,*
d *muscle soreness* may be present.
premonitory stage of the disease
eadache, back pains and painful
joints may be present and may not respond to any therapy until malaria is found and treated.[6,17,19,28,53,54,66,82]

nt Surg , 21, 665, 1939
Jour Am Med Assn , 122, 1177, 1943
23. BLOISE, N. L Arch de pediat d Uraguay, 4, 99, 1933
24 BLOOMBERG, M H New England Jour Med , 212, 1122, 1935
25 BLOOMGART, H L . Jour Am Med Assn , 111, 521, 1938
26 BLUMGART, H. L , and GILLIGAN, D R.: Med. Clin North America, 23, 1193, 1939
27 BOYD, C S Med Clin North America, 21, 1741, 1937
28 BROCKWAY, A Calif and West Med , 46, 174, 1937
29 BULKLEY, K Ann Surg , 59, 71, 1814
30 BUSH, F W , and BAILEY, F R Ann Int Med , 20, 619, 1944
31. CALDWELL, G A Jour Am Med Assn , 98, 37, 1932
32 CAMPBELL, S J Proc Staff Meet Mayo Clin , 13, 385, 1938
33 CAMPBELL, W , and GREENFIELD, E C South African Med Jour , 10, 545, 1936
34 CARNETT, J B · Ann Surg , 61, 456, 1915
35 CARTER, R A Am. Jour Roentgenol , 25, 715, 1931
36 CASEY, J F New England Jour Med , 215, 1282, 1936
37 CHEEVER, F S Quoted by Daniels *et al* [10]
38 CHICKERING, H T . Med Clin North America, 21, 1739, 1937.
39 CURTIS, S H New York State Med Jour , 32, 672, 1932
40 CUSHING, E H , and STOUT, A P Arch Surg , 12, 539, 1926
41 CUTLER, C W , JR Surg Clin North America, 18, 549, 1938
42 DALRYMPLE-CHAMPNEYS, W Lancet, 2, 1449, 1935
43 DANIELS, W B , SOLOMON, S , and JAQUETTE, W A , JR · Jour Am Med Assn., 123, 1, 1943
44 DARLEY, W , and GORDON, R W Jour Am Med Assn., 115, 2115, 1940
45 DAVIDSON, M Post-Grad Med Jour , 18, 166, 1942
46 DAWSON, M H , and BOOTS, R L Jour Am Med Assn , 113, 1162, 1939
47 DEKKERS, H J. N Acta med Scandinav , 108, 440, 1941
48 DELAPERSONNE, F · Des Arthrites Infectieuses (non-tuberculeuses), Paris, A Delahays and E Lecrosnier, 1886
49 DINDLE, J H , and FINLAND, M War Medicine, 2, 1, 1942
50 DOBSON, L , HOLMAN, E , and CUTTING, W Jour Am Med Assn , 116, 272, 1941
51 DUSTIN, C C Rhode Island Med Jour , 20, 73, 1937
52 ECKER, E E , KLEHN, A. O , and RECROFT, E W · Jour Am Med Assn , 118, 1296, 1942
53 FOGERS, G
54 Epstein, J
55 ERVIN, C
56 EVANS, A
57 EVANS, A C , ROBINSON, F H , and BAUMGARTNER, L Pub Health Rep , 53, 1507, 1938
58 FAGGE, C H Guy's Hosp Rep , 83, 441, 1933
59 FARRELL, E , LORDI, G H , and VOGEL, J · Arch Int Med , 64, 1, 1939
59a. FINLAND M , *et al* , Jour Am Med Assn , 138, 946, 1948
60 FISET, P E Canad Med Assn Jour , 47, 545, 1942
61 FISH, C A Personal communication
62 FLIPPIN, H F Med Rec , 149, 159, 1939
63 FOLEY, W B Proc Roy Soc Med , 34, 657, 1941
64 FOSHAY, L , and LEBLANC, T. J Jour Lab and Clin Med , 22, 1297, 1937
65 FOSTER, J B Texas State Jour Med , 28, 476, 1932
66 FOX, M J , and GILBERT, J Am Jour Med Sci , 208, 63, 1944
nt Surg , 18, 417, 1936

Black Water Fever.—This may be accompanied by lumbar pain and headache

VIRAL DISEASES

Dengue.—Prodromal symptoms may occur a few hours before the

hips and back, and may be agonizing, increased by the slightest active motion though unaffected by passive manipulation

Actually *the painful points are in the tendinous insertions about the joints rather than in the joints themselves.* With remission of fever, the pains often recede, perhaps to recur in less severe form as the temperature again mounts. These *limb pains referred to the joints occur in over half of the patients,* mild in some epidemics, intense enough in others to merit the name "*break-bone.*"

The joints usually show no sign of inflammation An occasional patient develops periarthritis of a knee or ankle which may give symptoms for months. The convalescent patient may continue to have pains for a month or more, worse in the morning and after a rest. Locomotion is difficult and walking is stilted, hence the name "dengue," a probable corruption of "dandy" The pains may persist in the hands and soles Eventually complete recovery usually occurs

Yellow Fever.—The onset is often sudden with severe headache and pain in the neck, back and limbs which may continue to increase for several days [14 48 53 54 66 82]

Rift Valley Fever.—This is found only in Africa, like certain other viral

RICKETTSIAL AND ALLIED DISEASES

Typhus.—The *onset* or stage of invasion is often accompanied by headache, pains in the limbs, especially in the calves,[14 22 56 60 66 79 83] and severe back pains During the *course* of the disease the patient often continues to be tormented by excruciating pains in the back and limbs These are among the commonest symptoms of the disease The pains may be migratory. There is usually no redness or swelling of the joints, but purulent joint effusion has been observed in one instance *During convalescence* pains in the legs and feet are common.

Rocky Mountain Spotted Fever and Tsutsugamushi—Pains similar to those of typhus are usually present Hyperesthesia may occur [1 84]

Q Fever.—This may cause pains in the back and legs.[19]

Trench Fever.—At the *onset* there is frequently headache, backache, pain in the b

shift or come

however, are

boring, milder in the daytime but so severe at night that the patient may keep all his companions awake with his groans They may regress during

71. GHORMLEY, R K., and DEACON, A. E. · Am. Jour. Roentgenol , 35, 740, 1936.
72. GOLDFAIN, E : Jour Oklahoma Med Assn , 31, 111, 1938
73 ——— South. Med Jour , 31, 325, 1938
74 ———. Jour Lab. and Clin. Med., 27, 168, 1941.
75. GOODYEAR, E S : Am Jour Dis Child , 61, 342, 1941.
76. GOULD, S E : Jour. Michigan Med Soc , 36, 637, 1937.
77. GOUNELLE, H , *et al* Bull et mém Soc méd. d hôp de Paris, 56, 82, 1941
78. GRACE, A. W . Arch. Dermat. and Syph , 48, 659, 1943.
79. GRACE, A W , and RAKE, G Arch. Dermat and Syph., 48, 619, 1943
80 GRAHAM, G Proc. Roy. Soc. Med , 13, 23, 1919
81. GREEN, F. B Texas State Jour Med , 32, 549, 1936
82 GREEN, M E., and FREYBERG, R. H. Am. Jour Med Sci , 201, 495, 1941.
83 GUTHRIE, K J . Arch Dis Child , 16, 269, 1941.
84. GWYN, N B · Phila Med Jour , 2, 1255, 1898
85 HAMILTON, G R Mil Surg , 83, 431, 1938.
86. HARDY, A V · Med Clin North America, 21, 1747, 1937
87 HARDY, A V , JORDAN, C F , and BORTS, I. H : Jour. Am. Med Assn , 107, 559, 1936
88 *HARDY, A V., JORDAN, C F, BORTS, I H , and HARDY, G C.: Pub Health Rep , 45, 2433, 1930*
89 HARMON, P. H · Jour Bone and Joint Surg , 24, 576, 1942.
90 HARRIS, H J New York State Jour Med , 37, 1295, 1937
91 ——— Jour Am. Med Assn , 122, 1204, 1943
92 [illegible]
93 [illegible] ıpson
94 [illegible] !2
95 [illegible]
96 [illegible]
97 HEFKE, H. W , and TURNER, V. C Jour. Bone and Joint Surg , 24, 857, 1942.
97a HEILMAN, F. R , Proc Staff Med Mayo Clin , 23, 569, 1948.
98 HENCH, P S Nelson's Loose Leaf Living Surg , New York, Thomas Nelson & *Sons, 3, 101, 1935*
99 ——— Ann Rheum Dis , 2, 172, 1941.
100 HENCH, P S , *et al* Ann Int Med , 9, 883, 1935.
101. ——— Ann Int. Med , 10, 754, 1936
102 ——— Ann. Int Med , 8, 1315, 1935.
103 ——— [illegible] 11 1080 1938
104 ——— [illegible]
105 ——— [illegible]
106 ——— [illegible]
107. HERRELL, [illegible] agents, Philadelphia, W. B Saunders Co , 1945
108 HERRELL, W E , and BROWN, A E Proc Staff Meet Mayo Clin , 14, 753, 1939
109 *HERRICK, W W Jour Am Med Assn , 71, 612, 1918*
110 ——— Bull New York Acad Med , 7, 487, 1931
111. HERRICK, W W , and PARKHURST, G M Am Jour Med Sci , 158, 473, 1919.
112. HILL, L. W , and LEVER, H S Jour Am Med Assn , 123, 9, 1943
[illegible] Acta chir Scandinav 84, 479, 1941
[illegible] Med , 238, 431, 1948
[illegible] 32, 1937.
[illegible] ›nimals, New York, The Common-
[illegible] ur. Trop Med , 17, 863, 1937.
[illegible] ʼnec and Obst , 60, 86, 1935
122 JACKMAN, J · Radiology, 40, 103, 1943
[illegible] de la Soc de cir. de hosp (Santiago, Chile), 12, 305, 1942
[illegible] er , 48, 179, 1938
[illegible] 935

apyrexial periods, and recur when the fever rises again. *Hyperesthesia is marked* and may be so severe that even the weight of the bed clothes is intolerable. *Joint pains occur especially in the knees and ankles.* In the chronic stage lumbar pain is likely to persist. Any thought of malingering is excluded by the presence of fever and the fact that pains are worse at night.

Oroya Fever.—In the *chronic mild type* of this disease and in the *febrile type* joint pains are common; in the latter type, headache and bone pains also occur. In the *Verruga peruana* stage there is an onset with rather severe *joint pains, especially in the knees, ankles and wrists, along with fever.* Constitutional symptoms subside with the appearance of skin lesions

VITAMIN DEFICIENCY DISEASES

Scurvy.—Pains in the legs are an outstanding symptom. Hemorrhage may extend from the calf or posterior thigh over the regions of the knee and ankle causing pain on motion of the joints. A knee and ankle may be edematous and painful without obvious hemorrhage. These cases of scurvy arthritis are found in a small proportion of cases in large epidemics[59,82] Occasionally the knee, ankle or hip may be acutely painful *without other local signs and pain sometimes subsides rapidly overnight*

In children the limbs may show the well-known subperiosteal hemorrhages with resultant swelling and tenderness and with violent pain on active or passive motion. *X*-ray shows the typical changes of periosteal detachment, more readily recognized after some calcium has been laid down; other findings may include subepiphyseal slipping or separation [illegible] less frequently in adults

[illegible] s in the limbs [illegible] umbar spine. The gait is often peculiar, the legs being separated and bent and the patient shuffling along with the feet barely raised from the ground. The gait *may suggest that of spastic paralysis.*[54 66 82]

Beriberi.—The calf muscles are sensitive to pressure. There may be burning of the feet which is made worse on walking. This, added to the weakness or actual paralysis due to neuritis makes walking difficult. [illegible]

[illegible]

and legs and may become completely bedridden [illegible] Typical pelvic trifoliate deform- [illegible] eformity of the legs may appear. [illegible] nshine in a pregnant or lactating [illegible] rrect diagnosis, especially if the symptoms are repeated and increased in severity in successive pregnancies.

FUNGUS DISEASES

Madura Foot.—*This chronic inflammatory disease usually attacks the foot* but may involve the hand, knee, thigh or buttock. There is marked swelling or deformity of the foot or hand with progressive disintegration of

127. JOURNE, H · Bull. Soc path expt, 27, 588, 1934
128. KATO, D, and LANE, C. R Lancet, 2, 77, 1937.
129. KAU, L S: Jour Clin Med, 6, 107, 1941
130. KEEFER, C S, MYERS, W K, and HOLMS, W F, JR Arch Int Med, 54, 8… 1934
131. KEEFER, C S, PARKER, F, JR, and MYERS, W K · Arch. Path, 18, 199, 19…
132. KENDRICK, J I Surg Clin North America, 17, 1475, 1937
133. KENT, J P Industr Med, 9, 368, 1940
134. KEY, J A, and LARGE, A M · Jour Bone and Joint Surg, 24, 281, 1912
135. KINI, M G, and KESAVASWAMY, P Antiseptic, 38, 169, 1941
136. KOBER, L R Southwestern Med, 21, 317, 1937
137. KOLLÁR, K Bratisl lek listy (Suppl 8), 19, 113, 1939
138. KRIDA, A Med Clin North America, 21, 1751, 1937
139. KRUSPE Dermat Wchnschr, 112, 477, 1941
140. KULOWSKI, J Nelson's Loose Leaf Living Surgery, New York, Thomas Nelson & Sons, 3, 47, 1935
141. ——— Surg, Gynec and Obst, 62, 759, 1936
142. ——— Medicina, Mexico, 21, 201, 1941
143. KULOWSKI, J, and VINKE, T H Jour Am Med Assn, 99, 1636, 1932
144. LABENDZINSKI, F Acta rheumatol, 3, 27, 1931
145. LAFFERTY, R H, and PHILLIPS, C C South Med Jour, 30, 595, 1937
146. LARSON, W P, and BARRON, M · Jour Infect Dis, 13, 429, 1913
147. LEAVITT, D G Northwest Med, 39, 169, 1940
148. L'EPISCOPO, J B Ann Surg 104, 289, 1936
149. LEVINE, N M, MYERS, J A, and LEGGETT, E A Jour Lancet, 57, 453, 1937
150. LEWIS, J T Brit Med Jour, 2, 1050, 1927
151. LONG, P H, *et al* Jour Am Med Assn, 115, 364, 1940
152. LORENTZ DE HAAS, A M Acta psychiat et neurol Copenhagen, 16, 405, 1941
153. MACKEE, G M, and ASTRACHAN, G D Jour Invest Dermat, 3, 409, 1940.
154. MARGOLIS, H M Proc Mayo Clin, 2, 121, 1927
155. MARSH, H Diseases of Joints and Spine, London, Cassell & Co, Ltd, 1895
156. MCCULLAGH, E P, and CLODFELTER, H M Ann Int Med, 10, 1509, 1937
157. MCILHENNY, P A New Orleans Med and Surg Jour, 84, 182, 1931
158. MCMASTER, P E, and GILFILLAN, C Jour Am Med Assn, 112, 1233, 1939
159. MEADS M *et al* North Carolina Med Jour, 9, 568, 1948
160. MEYER, M, and GALL, M B Jour Bone and Joint Surg, 15, 857, 1933
161. MEYERDING, H W Proc Staff Meet Mayo Clin, 10, 187, 1935
162. MILCH, H Jour Bone and Joint Surg, 24, 653, 1942
163. MILLER, J L Ann Int Med, 8, 570, 1934
164. MITCHELL, A G, and TRACHSLER, W H Jour Pediat, 11, 183, 1937
165. MORALES-OTERO, P, and GONZÁLEZ, L M Am Jour Med Sci, 199, 810, 1940
166. MURAKAMI, S Bull Naval Med Assn, Japan, 25, 87, 1936
167. MURRAY, R, PAINE, J F, and FINLAND, M New England Jour Med, 236, 701, 1947
168. MYERS, T H Am Jour Orthop Surg, 5, 180, 1907
169. NETTER and DURAND Quoted by Stevens, A A, The Practice of Medicine, Philadelphia, W B Saunders Company, 1926
170. NICHOLSON, J T Pennsylvania Med Jour, 43, 950, 1940
171. NIKOLSKIY, A P Klin Med, 13, 1223, 1935
172. O'DONOGHUE, A F Jour Bone and Joint Surg, 15, 506, 1933
173. OLDHAM, J B Lancet, 2, 576, 1937
174. OSLER, W Brit Med Jour, 1, 1521, 1899
175. O'LEARY, P A Canad Med Assn Jour, 48, 410, 1943
176. OVERTON, L M Proc Staff Meet Mayo Clin, 10, 85 1935
177. PAINE, T F *et al* Jour Bact, 66, 459, 1948
178. PAINTON, J F, and ULRICH, H J · Ann Int Med, 10, 1345, 1937
179. PALAGI, P Chir d org di movimento, 20, 31, 1934
180. PALMER, I South Med Jour, 31, 530, 1938
181. PARKER, F, JR, and HUDSON, N P Am Jour Path, 2, 357, 1926
182. PALL, J R, SALINGER, R, and ZUGER, B Jour Clin Invest, 13, 503, 1934
183. PERVASSO, D Brasil-medico, 53, 309, 1939
184. PETERSON, J C Jour Pediat, 7, 765, 1935.

all the tissues including the bones and joints, and with granulomatous ecognized

bones or joints with its gummatous or ulcerative lesions.

HELMINTH INFECTIONS

Filariasis.—In filariasis due to Wuchereria bancrofti, the filarial invasion is often accompanied by synovitis. Synovitis and arthritis may occur in the hip or knee; in some cases a purulent effusion is seen.[53 54 71] Surgical interference may be indicated Where the hip joint is involved, removal of the inflamed iliac glands may afford relief Fibrous ankylosis often results

Loiasis.—In loiasis the first symptom may be *transient swellings about the joints followed by stiffness.* The swellings may be painless or there may be aching and burning along with the swelling in an ankle, arm, hand or wrist[33,82]

Onchocerciasis.—In onchocerciasis acute arthritis has been described, with microfilaria in the joint fluid.[64 82,86] More commonly one finds small or large subcutaneous tumors (even up to the size of a pigeon's egg), often near the joints, in the popliteal space, knee, over the trochanter, in the axillary or shoulder region, about the elbow or over the vertebral column In the incipient stage these nodules may be quite painful, those near joints being the more easily inflamed If the nodule acts as a purulent focus, there may be pain and fistula formation with scarring and loss of function, the trochanters are a frequent site for such scarring

Dracontiasis.—In endemic areas, a bullous blister or sinus in the foot or leg is almost certainly due to the *guinea worm*, which often presents at the ankle, knee or foot.[43] There may be more than one sinus When the worm presents near a *joint*, that region becomes red, swollen and painful, and locomotion is difficult.[19 24 54 66 82] *Synovitis* and *arthritis* often develop, with a serous or purulent joint effusion This is usually sterile

Fibrotic changes and contraction of the tendons, as the Achilles tendon or the hamstrings, may occur, with or without bony ankylosis *Permanent deformities may result* from secondary bacterial infection, especially after long recumbency. Such bacterial infection of a joint may appear before there is any sinus tract *Arthritis may result from invasion of the joint by the worm itself or the nearby presence of a calcified worm*; a live worm burrowing deep between fascial planes may give pains without actual joint involvement.

Hookworm infestation is sometimes accompanied by joint pains

Other helminth infections that cause body pains, often more generalized, are:

Trichinosis, where the beginning of the period of larval migration into muscles is often associated with mild or severe muscle pains, sometimes with tenderness or periorbital edema. Pain on mastication may be a symptom. An erroneous diagnosis of "arthritis" or "rheumatism" is frequently made

Cysticercosis.—Cysticercosis may be accompanied by *myalgias* during the active life and development of the parasite.

185 Phalen, G S, Prickman, L E, and Krusen, F. H. Jour Am. Med Assn, 118, 859, 1942.
186 Pinelli, A · Pediatria, 35, 147, 1927
187 Place, E. H, and Sutton, L. E: Arch. Int Med, 54, 659, 1934.
188 Plisson and Brousse Lyon chir, 17, 705, 1920.
189. Ramond, L.· Presse méd, 49, 975, 1941
190 Regan, J. R Am Jour Surg, 31, 131, 1936.
191. Reiman, H A Jour Clin Invest, 14, 311, 1935
192 Reiss, O, and Kato, K. Am Jour. Dis. Child, 43, 365, 1932.
193 Reiter, H: Deutsch med Wchnschr., 42, 1535, 1916.
194 Rhoads, P S, and Afremow, M L: Ann. Int Med., 19, 60, 1943.
195 Riuz-Gijon, J Deutsch med. Wchnschr, 61, 1234, 1934
196 Robertson, R C · South. Med Jour, 30, 502, 1937
197 Rolfi, D V: Bol Soc de cir. de Rosario, 7, 47, 1940.
198 Rolleston, H Lancet, 1, 541, 1919
199 Rolly Quoted by Hench Ref No 111
200 Rosenberg, E. F, Dockerty, M B, and Meyerding, H W.· Arch Int Med, 69, 238, 1942.
201 Rule, C, and Grollman, A Ann Int Med, 20, 63, 1944
202 Sainton, P Lancet, 1, 1080, 1919.
203 Salvesen, H A. Norsk. mag f. lægevidensk., 98, 953, 1937.
204 Samokhin, G I · Vrach delo, 22, 697, 1940
[illegible]
210 Schein, A J. Arch Int Med, 62, 963, 1938
211 Schein, A J, and Arkin, A M Jour. Bone and Jour. Surg, 24, 396, 1942.
212 Schottmüller, H. Dermat Wchnschr. (Suppl), 58, 77, 1914
213 Sevier, C E Colorado Med, 29, 251, 1932
214 Sharpe, J C Ann Int Med., 9, 1431, 1936
215 Shattuck, G [illegible]
216 Shuller, E [illegible]
217 Silver, D [illegible]
218 Simmons, J S [illegible]
219. Simpson, W [illegible]
220 Simpson, W. M., and Frazier, E. Jour Am Med Assn, 93, 1958, 1929.
221. Slowick, F. A: New England Jour Med, 212, 672, 1935
222 Smith, H. W, Thomas, L, Dingle, J H, and Finland, M: Ann Int. Med, 20, 12, 1944
222a Smith, R M, Jour Bact, 55, 425, 1948
223 Snyder, C H · Jour Michigan Med Soc, 34, 224, 1935
224 Sokolova, Y. V, and Khondu, A A, and Kolegaeva, A I · Vestnik khir, 44, 30, 1936.
225 Solomon, W M, and Tuchewicz, H. R Jour Bone and Joint Surg, 20, 1061, 1938
[illegible] d Sci,
230 Strong, P. S, and Hollander, J L Bull of the U S Army Med Dept, 78, 68, 1944
231. [illegible] Bone and Joint Surg, 20, 473, 1938
232. [illegible]
233 [illegible] Am Jour
234. [illegible] Surg, 43, 655, 1939.
235. Traut, E. F., and Logan, C. E.· Jour. Am Med Assn., 111, 1092, 1938
236 Tur, A. F, and Gartoch, O. O · Ztschr f. Kinderh, 56, 696, 1934.

In schistosomiasis there may be toxic symptoms at the end of the incubation period including persistent headache and pains in the back and limbs.

There are several *disease entities of unknown etiology* where joint involvement may be present or be simulated:

Tropical ulcer usually attacks the lower part of the leg or the foot. An [illegible] Coast, and usually attacks the quadriceps or gluteal muscles.[25,82] It must be distinguished from cellulitis and septic arthritis.

In **Ainhum** a circular groove appears at the base of one or both little toes, less often the fourth toes, deepens gradually and in the course of several years proceeds to the severance of the toe, usually without ulceration or pain.[51,54,82] X-ray shows absorption of the ungual tufts and of other parts of the bones, especially in the neighborhood of the constriction.

Big Heel, occurring on the African Gold Coast, begins with tenderness in one or both heels with enlargement of the os calcis, resulting in marked pain on walking.

Lathyrism.—Lathyrism, in India, Africa and elsewhere, has symptoms similar to spastic paralysis with ataxia. At the onset there are pains in the back and weakness of the legs.[54,80,82] The disease is thought to be due to eating bread made of flour from the chick-pea, Lathyrus sativus, or possibly a contaminating weed, Vicia sativa Deleterious effects are evident only when a considerable amount is consumed.

Epidemic Dropsy, somewhat similar to beriberi, thought to be caused by toxic properties of oils used in the diet, has as its prominent symptoms aches in joints, muscles and bones.

Drugs.—Various drugs used in the treatment of certain tropical diseases may, on occasion, give rise to generalized pains or pains in muscles or joints:

The milder symptoms of **aspidium** poisoning include muscle cramps.[80]

Several of the drugs used in treating African trypanosomiasis have frequently caused headache and generalized body pains, *viz:* **antrypol** (the English equivalent of *Bayer 205*,[18,27,82]), **tryparsamide**, or the combined treatment of antrypol followed by tryparsamide.[52] With these drugs the reactions are usually mild but sometimes severe enough to necessitate discontinuance of treatment.

In the treatment of kala-azar or oriental sore with **antimony** preparations intravenously, immediate reactions often include a feeling of *muscle stiffness.* Toward the end of a course of therapy *severe pains in joints and muscles with cramps in the calves* may occur four to five hours after an injection and may last as long as twelve hours.[54,74,82] Such pains must be regarded as danger signals. They are less common with the less toxic pentavalent antimony compounds than with the trivalent sodium or potassium antimony tartrate.

In this connection one must mention the pain in the legs so bitterly complained of by many *opium addicts when the drug is withdrawn.*[80] Such pain sometimes persists for days or weeks after all other symptoms of craving have disappeared.

237 TWEDDELL, J. T., and SCHLOTZHAUER, K B: Canad Med Assn Jour, 30, 653, 1934.
238 VEAL, J R: New Orleans Med and Surg Jour, 87, 549, 1935
239 VREELAND, C E Jour Michigan State Med Soc, 35, 632, 1936
240 WAGHELSTEIN, J M Jour Am Med Assn, 111, 2172, 1938
241 WAINWRIGHT, C. W.: South. Med Jour, 30, 600, 1937
242 WEAVER, J B, and SHERWOOD, L Jour Am Med Assn, 105, 1188, 1935
243 WEISMAN, J I: New England Jour Med, 216, 977, 1937
244 WELT, S, ROSENTHAL, N, and OPPENHEIMER, B S Jour Am Med Assn, 92, 637, 1929
245 WILLIAMS, R H Arch Int Med, 68, 80, 1941
246 WILLNER, P, PLACE, E H, and SUTTON, L. E, JR. Med and Surg Jour, 194, 285, 1926
247 WILSON, D E [illegible]
248 WINANS, [illegible]
249 WISE, B [illegible]
250 WITZEL [illegible]
250a WONG, S C and SANDERS, M: Jour Am Med Assn, 138, 426, 1948
251 WOODWARD, L R Jour Iowa Med Soc, 27, 609, 1937

The sulfonamides are finding their place of usefulness in treatment in certain tropical diseases and the occasional appearance of joint pains (sulfatoxic arthritis) due to their use must be mentioned

BIBLIOGRAPHY

1 AHLM, C E, and LIPSHUTZ, J Jour Am Med Assn, 124, 1095, 1944
2 ASHBURN, P M, and CRAIG, C F Jour Infect Dis, 4, 440, 1907
3 BAZAN, F, and MAGGI, R. Arch argent. de pediat, 12, 376, 1939
4. BEESON, P B. Lancet, 2, 67, 1941
5 BENJAFIELD, J D, and HALLEY, G S Lancet, 2, 616, 1938
6 BIRKS, P H Brit Med Jour, 1, 784, 1943
7. BONNIN, N J, and KAY, H B Med Jour Australia, 2, 380, 1943
8 BRAHMACHARI, U, and BASU, C C Jour Trop Med and Hyg, 45, 81, 1942
9 BROCKLEBANK, J A Brit J Radiol, J 16, 221, 1943
10 BROWN, T McP, and NUNEMAKER, C Bull Johns Hopkins Hosp, 70,201, 1942
11 BULMER, E, and PRIEST, W M Jour Roy Army Med Corps, 79, 277, 1942
12 CARPENTER, C M Med Clin North America, 27, 698, 1943
13 CARTER, R A Am Jour Roentgenol, 35, 715, 1931
14 CECIL, R L A Textbook of Medicine, 6th ed, Philadelphia, W B Saunders Company, 1942

1943

43

19 CRAIG, C F, and FAUST, E C Clinical Parasitology, 5th ed, Philadelphia, Lea & Febiger, 1953
20 CUÉLLAR, P I V Bol Ofic san panam, 20, 807, 1941
21 D'ANTONI, J S Clinics, 2, 936, 1943
22 DYER, R E Jour Am Med Assn, 124, 1165, 1944
23 EARLE, K V Jour Trop Med, 44, 88, 1941
24 ELLIOTT, M Trans Roy Soc Trop Med and Hyg, 35, 291, 1942
25 ERASMUS, J
26 FAGET, G H 42
27 FAIN, A
28 FEREAUD, C
29 GOLDMAN, C H, and SMITH, S J Brit Jour Radiol, 16, 234, 1943
30 GOUNELLE, H, *et al* Bull et mém Soc méd d hôp de Paris, 56, 82, 1941
31 GRAHAM, G Proc Roy Soc Med, 13, 23, 1919
32 GREEN, M E, and FREYBERG, R H Am Jour Med Sci, 201, 495, 1941
33 GUY, W H, COHEN, M, and JACOB, F M Arch Dermat and Syph, 47, 763, 1943
34 HALLIDAY, J H, and HORAN, J P Med Jour Australia, 2, 293, 1943
35 HARDY, A V, *et al* Pub Health Rep, 45, 2413, 1930
36 HARRIS, L Med Jour Australia, 1, 516, 1944
37 HEALEY, C E Jour Am Med Assn, 108, 628, 1937
38 HENCH, P S, *et al* Ann Int Med, 12, 1005, 1939
39 ——— *Ibid*, 28, 320, 1948
40 HIDDE, F G Essential Technical Medical Data, U S Army A P O, 921, 1943
41 HUDSON, E H Trans Roy Soc Trop Med and Hyg, 28, 511, 1935
42 JACOBI, J Munchen med Wehnschr, 89, 615, 1942
43 JOURNE, H Bull Soc path expt, 27, 588, 1934

50 LOPEZ, P. Rev d Inst salub y enferm trop, 3, 41, 1942
51. DELORIMIER, A A The Arthropathies, a Handbook of Roentgen D Chicago, Year Book Pub Company, 1943.

CHAPTER 48

ARTHRITIS IN TROPICAL DISEASES

(Originally Written By JULIA MORGAN, M.D., AND BERNARD I. COMROE, M.D.)

Revised By VICTOR G BALBONI, M D.

Arthritis is a well-known complication in a few tropical diseases, notably *bacillary dysentery, undulant fever, the fungus infections,* coccidioidomycosis, madura foot, and in *filariasis* due to Wuchereria bancrofti. *Arthritis may occur occasionally,* incidentally or as a result of extension by contiguity from the more typical lesions elsewhere, in a number of other diseases: the spirochetal diseases *yaws, bejel, relapsing fever and rat-bite fever;* the fungus diseases, *sporotrichosis and histoplasmosis;* the nematode infections, *onchocerciasis and dracontiasis;* in *leprosy, smallpox, scurvy* and in *tropical ulcer.*

Arthralgias are common and *occur in* the rickettsial group in *typhus,*

[illegible]

loa; in onchocerciasis and *dracontiasis;* in *hookworm infection;* also in scurvy, epidemic dropsy, *malaria,* African trypanosomiasis, and sometimes after antimony injections.

Pains in the limbs are described in a still larger group of diseases, frequently at the onset of the fever, and often associated with headache, backache or generalized body pains. In three groups of diseases, the spirochetal group, the rickettsial diseases and the viral diseases, the pains are *severe and so regularly present as to be a helpful diagnostic aid.* Most of the other diseases and conditions described may at times give pain in the limbs of greater or lesser degree

Diseases which *seldom or never produce arthritis, joint pains* or other pains likely to be confused with joint pains are *amebiasis* and the *leishmania* infections.

Rheumatic diseases may differ somewhat as to their occurrence in *temperate or in tropical regions* *A number of observers find that* although rheumatic fever may occur with its usual frequency among European residents in many tropical areas, in the native races in the tropics both rheumatic fever and rheumatoid arthritis are comparatively uncommon. Among Chinese patients in North China the situation with regard to arthritis and allied conditions seems to be analogous to that in our own southern states. Rheumatic heart disease with mitral stenosis is commonly seen, though acute attacks of rheumatic fever are observed infrequently and a history of such an illness can be elicited in very few of the cardiac patients Some experienced workers in India, Malaya, South

52 LOURIE, E M · Ann Trop Med, 36, 113, 1942
53 *MACARTHUR*, W. P. *Memoranda on Medical Diseases in Tropical and Subtropical Areas*, War Office (British), 1st American ed, Brooklyn, N. Y., Chemical Publishing Company, 1942.
54 MANSON-BAHR, P. H · Manson's Tropical Diseases, 11th ed, Baltimore, Williams & Wilkins Company, 1940 and 1942.
55. MCILHENNY, P. A. New Orleans Med and Surg Jour, 84, 182, 1931
56. MENDOZA, L. Mem. d V Congr Med Centroamericano, San Salvador, Nov, 1938.
57. MEYER, K F. Med Clin *North America*, 27, 745, *1943*
58 MEYER, M, and GALL, M B: Jour Bone and Joint Surg, 15, 857, 1935
59. MORGAN, J, and GAULT, A S · Chinese Med Jour, 60, 141, 1941
60 MRUGOWSKY, J. Med. Klin, 38, 193, 221, 1942.
57. MUIR, E Leprosy Rev, 14, 49 and 51, 1943
62 MURSKAMI, S Bull Naval Med Assn Japan, 25, 87, 1936
63 NIKELSKIY, A P. Klin Med, 13, 1223, 1935
60. PHALEN, G. S, PRICKMAN, L E, and KRUSEN, F. H.: Jour. Am Med Assn., 118, 859, 1942
61 PROUK, K J Geneesk tijdschr v Nederl-Indië, 81, 1403, 1941.
62. ROGERS, L, and MEGAW, J W D. Tropical Medicine, 4th ed, London, J & A Churchill, 1942
63 ROMERO ESCACENA, G. Rev clin espan, 2, 349, 1941
64 ROSENBERG, E F, DOCKERTY, M B, and MEYERDING, H W.· Arch Int Med., 69, 238, 1942.
69 ROST, G. S Radiology, 38, 320, 1942
70 [illegible]
71 [illegible]
72. [illegible]
73 [illegible]
74 [illegible] 942.
75 [illegible]
76 [illegible]
77 SIMPSON, W. M, and FRAZIER, E Jour Am Med Assn, 93, 1958, 1929
78 SMITH, C E. Med. Clin North America, 27, 790, 1943
79 SNAPPER, I Chinese Lessons to Western Medicine, New York, Interscience, 1941
80 SOLLMAN, T A Manual of Pharmacology, 6th ed, Philadelphia, W. B. Saunders Company, 1943
81 STRONG, R P: Med Clin North America, 27, 734, 1943
82. ———: Stitt's Diagnosis, Prevention and Treatment of Tropical Diseases, Philadelphia, The Blakiston Company, 1944
83 SYLLA, A Med Klin, 38, 72, 1942
84 TAYLOR, R G. [illegible] 10, 551, 1923
85 *TOPPING*, N H · [illegible]
86 *VAN DEN BERGHE* [illegible] 67, 1941
87. VON BORMANN, F [illegible]
88 WEPLER, W Be[illegible]
89 WEST, R. F · Med Jour. Australia, 1, 344, 1943
90 WINDORFER, A · Deutsch med Wchnschr, 68, 1144, 1942.
91 YONIS, Z Harefuah, 24, 134, 1943

China and Central Africa report never having seen rheumatic fever or endocarditis in a life-long experience. Chorea is reported to be rare among children in East Africa However, among natives in the Gulf of Guinea region, "rheumatic arthritis" is said to be the commonest joint disease Gonorrheal arthritis is commonly reported in Ceylon, India and China, through actual statistics are incomplete and difficult to obtain. Tuberculosis of joints is more common in the tropics probably because tropical regions are largely unsanitated and the incidence of tuberculosis there is, in general, very high.

In addition to the forms of arthritis seen in temperate climates, the tropics present a variety of joint affections associated with the distinctively tropical diseases, as well as bodily aches and pains of various sorts occurring in connection with such diseases which must be differentiated from true arthritis

Some of the diseases discussed are found in the tropics only, others are exotic but not exclusively tropical while many occur in our own country but are commoner in tropical regions. Though they might be included in this last category, tuberculosis, typhoid and the venereal diseases are discussed in previous sections.

BACTERIAL DISEASES

Bacillary Dysentery.[7 11,21 53 54,56 82,89]—Arthritis is a well-known complication of bacillary dysentery.[30] It occurs in approximately 3 per cent of the cases, and is *more common in Shiga infections.*[85] It usually appears as the acute symptoms subside or within the first three to four weeks, but may develop up to three months or more[5] As a rule, the *large joints are affected*, especially the knees, on one or both sides. One or multiple joints may be involved.[52 53,70] Next in order of frequency are the ankles, elbows, fingers, shoulders and wrists. Some observers place hip joints next in order to the ankles

At the onset there is fever and pain, followed by effusion into the joint cavity of straw-colored, slightly viscid and usually sterile fluid. *Shigella organisms are occasionally cultivated from the joint fluid* and agglutinins are present, sometimes in higher titer than in the blood. Joint symptoms persist for a few days to six months but eventually there is complete recovery in practically every case[8 21] Suppuration and resultant ankylosis have occasionally been noted[37]

In the diagnosis one must beware of confusing dysenteric arthritis with that due to recent serum therapy. *This complication of dysentery is not* prevented by sulfonamides Various authors have discussed the rôle of dysentery in Reiter's disease[15 44] (See Chapter on Reiter's Syndrome p 795)

[illegible]

disease there may be stiffness and numbness of the extremities and *pain and swelling of hands and feet*

PART IX

MISCELLANEOUS (RARE) CONDITIONS OF OBSCURE ETIOLOGY SOMETIMES MISTAKEN FOR RHEUMATOID ARTHRITIS

CHAPTER 49

HEMOPHILIC ARTHRITIS

By MORRIS A BOWIE, M D.

HEMOPHILIA is an hereditary disorder of the blood transmitted as a sex-linked mendelian recessive from the male through an unaffected daughter ("conductor") to a grandson The sons of an affected male are normal themselves and cannot transmit the defect to any of their descend-



FIG. 262 —Chronic hemophilic arthritis of the knee following intermittent attacks of hemarthrosis for twenty years in this hemophiliac aged thirty-four

Chaulmoogra oil has been tried in the treatment of rheumatoid arthritis partly because of the supposed immunity of lepers to arthritis.[55] In a recent analysis it was found that 4.5 per cent of 425 patients at the Carville, Louisiana, Leprosarium had had a previous diagnosis of "arthritis," "rheumatism" or acute rheumatic fever.

Contractions and distortions of fingers and toes may occur fairly early in leprosy. Later, as infiltration of nerves proceeds, *trophic disorders of bones and joints* as well as of muscles become apparent. *Contractures* of the hand are likely to assume the main-en-griffe appearance, with function perhaps little impaired. Sometimes only the fourth and fifth fingers show contracture. In the feet, contractures are less common. There may be [illegible]

The pseudo-arthritis of lepers is mainly an osseous destruction; disintegration of the epiphyses and diaphyses follows destruction changes in the phalanges and metacarpal and metatarsal bones due to trophic changes. Eventually there may be spontaneous amputation of fingers or toes in whole or in part Even without ulceration, the bones of the digits may become rarefied and absorbed so that bones and joints disappear and the finger n[illegible] With certain joints, wrists a[illegible] *process similar to a Charcot* [illegible] isorganization of cartilage and bone.

Bubonic Plague.—Prodromal pains occur in the back and limbs. As the mental state deteriorates, the pains are less evident. The site of the [illegible] before the bubo appears. *With* [illegible] velops [illegible] ckache and generalized body aches [42,54,82] Pains may be fleeting or they may persist two to four weeks They may settle in the shin bones. In the chronic disease localized pains may recur at intervals for as long as a year.

Cholera.—*Serofibrinous synovitis* has been reported; general aches and pains may occur in the muscles of the extremities, then spreading upward and increasing in intensity. Similar symptoms, usually in milder degree, may occur in severe acute dehydration from any cause, as in acute bacillary dysentery and acute food poisoning.[66,82]

Epidemic Tropical Acute Polyarthritis.—(*"Foxhole Arthritis"; "Bougainville Rheumatism*—In September 1942, a heretofore undescribed type of rheumatism appeared among Australian soldiers in the Darwin-Adelaide River area of the northern territory of Australia It was characterized by acute polyarthritis, mild fever, transient rash, and lymphadenopathy. Hidde studied 29 cases at the 135 Medical Regiment Hospital and reported them in a "secret paper" to the Surgeon General of The United States [illegible] northern terri-

active substance can be precipitated from plasma by acid, and this substance is not present in hemophilic plasma; this active material is largely concentrated in fraction I and subfraction II of fraction III of Cohn [30,31,50,56]

sudden with severe pain, warmth and swelling, and fixation in flexion

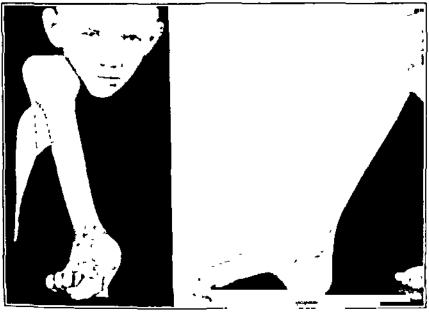


FIG 264 —Acute hemarthrosis of an ankle in a hemophiliac (Key, courtesy of Ann Surg)

Bleeding may be directly into the joint cavity or into either the epiphysis or diaphysis Usually one joint is involved at a time, although others may be subsequently attacked Recurrences are common In Thomas' series

cent), elbows (53 per cent), hips (16 per cent), fingers (15 per cent), wrist (5 per cent), spine (3 per cent), and toes (2 per cent) The age range was from early infancy to 65 years Involvement of the shoulders was rare.

The acute phase may persist for a few days to weeks Absorption of blood is often incomplete, retained blood causes a chronic synovitis with resulting pain, swelling, and tenderness Acute hemarthroses may be repeated, the

Surgery is to be avoided if at all possible, major surgery is attended by [illegible] very high mortality rate in hemophiliacs If unavoidable, pre-operativ[illegible] preparation with blood, plasma, or fraction I is indicated. Use of the electro[illegible] cautery has proved helpful [19] Maggots have been used to clean up wound[illegible] produced by necrosis following large hematomata [43]

Regular dental prophylaxis and care are of paramount importance Cav[illegible] ities can usually be filled without inducing any bleeding, but extractions may be attended by severe bleeding. Special techniques have been employed to partially free the gingival edges beginning a week or so prior to actual extraction [15] Blood or plasma should be administered an hour or so before operation Local anesthesia is applied with a very fine needle at the free cuff margin The extraction site should be cleaned, packed with dried thrombin onto which oxidized cellulose is packed, and the previously made denture is then fitted and allowed to remain in place for a matter of days. Suturing of the gums is contraindicated

The prognosis of hemophilia has improved since the use of blood and plasma, but the life expectancy is much shortened [9,59] In the past the greatest number of deaths followed exsanguination after surgical procedures such as circumcision, dental extraction, tonsillectomy, or incision of a hematoma

BIBLIOGRAPHY

1 ALEXANDER, B Jour Am Med Assn, 138, 174, 1948
2 ——— Jour Clin Invest, 27, 98, 1948
3 ——— Ibid, 28, 1511, 1949 (Part II)
4 ——— Am Jour Physiol, 163, 148, 1950
5 ALEXANDER, B, GOLDSTEIN, R, and LANDWEHR, G Jour Clin Invest, 29, 881, 1950
6 ——— Ibid, 30, 252, 1951
7 BAKER, G A, and GIBSON, P C Lancet, 1, 428, 1936
8 BARNETT, B Proc Royal Soc Med 28, 1469, 1935
9 BIRCH, C L "Hemophilia Clinical and Genetic Aspects," Univ of Illinois Bull, 34, No 15, Urbana, Univ of Illinois, 1939 (Review)
10 BRINKHOUS, K M Am Jour Med Sci, 198, 509, 1939
11 ——— Proc Soc Exper Biol and Med, 66, 117, 1947
12 BULS, C E P Acta radiol, 16, 503, 1935
13 CAFFEY, J, and SCHLESINGER E R Jour Pediatrics, 16, 549, 1940
14 DOUB, H P, and DAVIDSON, L C Radiology, 6, 217, 1926
15 DAVIDSON, E C, EPSTEIN, R D, MILLER, G F, and TAYLOR, F H L Blood, 4, 97, 1949
16 DAVIDSON, E C, and LEVENSON, S M Jour Am Med Assn, 128, 656, 1945
17 ENGELS, H Fortschr a d geb Roentgenstrahlen, 25, 197, 1917
18 FERGUSON, J H Proc Soc Exper Biol and Med, 67, 228, 1948
19 FIROR, W M, and WOODHALL, B Bull Johns Hopkins Hosp, 59, 237, 1936
20 FREUND, E Virchows Arch, 256, 158, 1925
21 GIACCAI, L Ann Radiol diag, 19, 325, 1947
22 GIRORDET, P Ann Pediat, 170, 169, 1948
23 GRAHAM, J B, BUCKWALTER, J A, HARTLEY, L J, and BRINKHOUS, K M Jour Exper Med, 90, 97, 1949
24 ——— Am J Physiol, 164, 710, 1951
25 GHORMLEY, R K, and CLEGG, R S Jour Bone and Joint Surg, 30-A, 589, 1948
26 HOWELL, W H Bull New York Acad Med, 15, 3, 1939
27 JOHNSON, J B Jour Am Med Assn, 118, 799, 1942
28 KEEFER, C S, and MYERS, W K Med Clin North America, 16, 929, 1933
29 KEY, J A Ann Surg, 95, 198, 1932
30 LOZNER, E. L, KARK, R, and TAYLOR, F H L Jour Clin Invest, 18, 603, 1939

ants, the daughters of an affected male, though not afflicted with any hemorrhagic diathesis, can transmit the trait to half of their sons, and, as a recessive characteristic to half of their daughters.[9,39] Its fundamental anomaly is a markedly decreased coagulation of the blood. Agreement among hematologists as to the exact mechanism on which delayed coagulation depends has not been reached, but the weight of evidence suggests that a deficiency or defect is present in the Phase I precursors of thromboplastin. Quick has concluded there is a deficit in the plasma factor (thromboplastino-



FIG. 263—Same patient as preceding figure Note marked narrowing of joint space (indicating destruction and wearing down of articular cartilage).

not reduced in number[5,6,40,41]. plasma or of plasma filtered free of platelets produces rapid shortening of the coagulation time, but transfusion of serum does not.[1,2,3,4,18,23,30,36] The

injections of any substance are best avoided since hematomata may result. 200 to 600 milligrams of *Cohn's fraction I of plasma* dissolved in a small amount of physiological saline solution are equivalent to about 80 cc. of fresh plasma or 100 cc. of whole blood.[37,59] The effect of this material lasts six to twelve hours; in order to determine the duration of action in acutely ill patients, the coagulation and prothrombin comsumption times should be determined several times in the first twelve hours after injection. Some patients have been maintained on regular weekly injections of plasma; this may not be necessary in more than a few since hemophiliacs often remain symptomless for long periods of time. Furthermore, a refractory state may develop[41,45] to these substances if used repeatedly.

Treatment of Hemophilic Arthritis

ACUTE HEMARTHROSIS.

1. *Splint* the affected joint with the patient at complete bed rest until pain and distension decrease
2. Apply ice bags initially and for a continued period if bleeding persists
3. Aspirate only if distension is very painful, use a fine bore needle (24 or 25 gauge).
4. Agents to improve coagulation defect:
   - *a* Transfusion of fresh whole blood (250 to 500 cc) or of fresh plasma 100 to 250 cc.)[15 37]
   - *b* Or transfusion with lyophilyzed plasma (100 to 250 cc)[37] or human globulin[37]
   - *c* Determine the coagulation time and prothrombin consumption time at the end of 30 minutes and after 8 hours Repeat the transfusions as often as may be indicated
   - *d* For local hemostasis use rabbit thrombin,[31] globulin from beef plasma,[46] or thromboplastin prepared from lung[25] Russell viper or Fer-de-lance venom have been recommended.[7,8,34,42]
     - (1) Clean wound carefully so that hemostatic will reach bleeding surface.

CHRONIC HEMOPHILIC ARTHRITIS

1. Correct deformities by conservative measures such as traction, wedging, mechanical appliances
2. Use whole blood, fresh plasma, or Cohn's plasma fraction I if fresh bleeding develops.
3. Avoid manipulation[28 59]
4. Employ hydrotherapy and passive motion when permissible
5. Protect joint with elastic bandaging reinforced with felt side pieces

Local hemostasis can be obtained by employing rabbit thrombin[31] or similar pseudo-globulin fractions prepared from beef or human blood [illegible] may have some value.[25] Russell viper [illegible] effect. The wound [illegible] is in direct contact [illegible] e proved effective [illegible] when given orally.

active substance can be precipitated from plasma by acid, and this sub-

sudden with severe pain, warmth and swelling, and fixation in flexion



Fig 264 —Acute hemarthrosis of an ankle in a hemophiliac (Key, courtesy of Ann Surg)

Bleeding may be directly into the joint cavity or into either the epiphysis or diaphysis Usually one joint is involved at a time, although others may be subsequently attacked. Recurrences are common In Thomas' series of 98 cases of hemophilia, arthritis developed in 79 per cent, and more than one-half of these incurred permanent deformity [81] In the *order of frequency joint involvement occurred* in the knee in 68 per cent, the ankles (56 per cent), elbows (53 per cent), hips (16 per cent), fingers (15 per cent), wrist (5 per cent), spine (3 per cent); and toes (2 per cent) The age range was from early infancy to 65 years Involvement of the shoulders was rare.

The acute phase may persist for a few days to weeks. Absorption of blood is often incomplete, retained blood causes a chronic synovitis with resulting pain, swelling, and tenderness. Acute hemarthroses may be repeated, the

CHAPTER 50

SARCOIDOSIS

(Besnier-Boeck-Schaumann Disease)

By John Lansbury, M.D.

Sarcoidosis is included in this section because it may present joint manifestations such as pain and swelling which bear a superficial resemblance to rheumatoid arthritis. To date it can only be classified as a disease of unknown etiology, although an infectious basis such as tuberculosis, has been suspected but never proved It is a not uncommon disease which produces characteristic lesions distinct from other infectious granulomas and which may affect almost any organ of the body, although there is a predilection for certain sites. This has given rise to separate clinical descriptions in the various special fields of medicine under such names as uveoparotid fever, osteitis tuberculosa multiplex cystoides, benign lymphogranulomatosis, lupus pernio, Boeck's sarcoid, etc Only in recent years have these syndromes been recognized as local manifestations of the same [illegible]

alt [illegible]

wl [illegible]

sex. The course is chronic and benign often extending over many years with remissions and relapses and generally eventual spontaneous recovery Death occurs in about 5 per cent of cases due to mechanical interference in [illegible]

tures Skin reactions to old tuberculin are minimal or negative, but become positive if ordinary tuberculosis complicates the disease, as it often does These unusual features have given rise to the theory that sarcoidosis is an 'anergic' phase of tuberculosis, but, against this one may cite a similar lack [illegible]

[illegible]

giant cell but without central necrosis or caseation. These lesions have [illegible]

capsule becomes greatly thickened, and punched-out areas due to subchrondral hemorrhages may appear. Cavitation may develop from intraosseus hemorrhage.[19,23] [illegible] rare, but fibrous ankylosis begins relatively early.



FIG 265 —Marked contractures of both knees in a severe hemophiliac seven years of age (Key, courtesy of Ann Surg)

The terminal stage leading to hemophilic arthritis may be heralded by subluxations or fibrous and bony ankylosis. In the smaller articulations complete destruction of the joint pattern and even of the bones is not infrequent. Both atrophy and proliferation of bone occur, depending on interference with the nutrition of the bone or excessive flow of blood to the growing epiphyses[13,35]

X-ray films of an acute hemarthrosis show a bulged out capsule and widened joint space.[1,12,20] Increased density due to the blood is readily apparent. Subsequently, opacities in the soft tissues may be visible due to the deposition of iron pigment in the organizing hematomata[2,14,35,38] Distortion of the joint cleft due to the irregular destruction of cartilage together with rarefactions, cysts, and cavities, and eroded subchrondral

31. LOZNER, E L, MACDONALD, H., FINLAND, M., and TAYLOR, F. H. L: Am Jour. Med Sci, 202, 593, 1941.
32 ——— *Ibid*, 205, 538, 1943.
33 MACFARLANE, R. G. St Bartholomew's Hosp Rep, 68, 229, 1935.
34 ——— Jour. Clin Path, 1, 113, 1948
35 MCDONALD, E J, and LOZNER, E L. Am Jour. Roentgenol, 49, 405, 1943
36. MILSTONE, J. H. Blood, 4, 1290, 1949.
37 MINOT, G R, DAVIDSON, C S., LEWIS, J H, TAGNON, H J, and TAYLOR, F. H. L.: Jour Clin Invest, 24, 704, 1945
38 NEWCOMER, N. B. Radiology, 32, 573, 1939
39 NOLF, P. Medicine, 17, 381, 1938 (Review)
40 ORR, W F., and GRAY, M. E Am Jour. Physiol., 163, 148, 1950
41 OWREN, P. A: Lancet, 1, 446, 1947.
42 PECK, S M, CRIMMINS, M I, and ERF, L A: Proc. Soc. Exper. Biol and Med, 32, 1525, 1935.
43 POHLE, F. J., and MADDOCK, S Jour Am Med Assn, 109, 2055, 1937
44 POHLE, F. J, and TAYLOR, F H L Jour. Clin Invest, 16, 741, 1937.
45. ———. *Ibid*, 17, 677, 1938
46 ———. *Ibid*, 17, 779, 1938.
47. QUICK, A J. "The Hemorrhagic Diseases and the Physiology of Hemostasis Springfield, Illinois, Charles C Thomas, 1942.
48. REINECKE, and WOHLWILL, F Arch f klin Chir, 154, 425, 1929.
49. SOLIS-COHEN, L, and LEVINE, S Am Jour. Roentgenol, 31, 487, 1934
50 TAYLOR, F H L· Am Pract, 2, 811, 1948
51 THOMAS, H B Jour Bone and Joint Surg, 18, 140, 1936.
52 TOCANTINS, L M Am Jour Physiol, 139, 265, 1943
53 ——— *Ibid*, 143, 67, 1945
54. ——— Blood, 1, 156, 1946
55. TOCANTINS, L M, CARROLL, R. T, and HOLBURN, R. H.: Blood, 6, 720, 1951
56 WARE, A G, and SEEGERS, W H Am Jour. Physiol, 152, 567, 1948
57. ——— Blood, 5, 303, 1950
58 WHITE, C E Jour. Oklahoma Med Assn., 25, 304, 1932.
59 WINTROBE, M M.· "Clinical Hematology," 3rd ed., Philadelphia, Lea & Febiger, 1951
60 WRIGHT, C S Jour Lab and Clin Med., 33, 708, 1948

areas appear in more advanced cases.[14,17,38,43] Accelerated epiphyseal maturation may be seen early in hemophilia if the child has had repeated hemarthroses.[13,35] In 15 adults with hemophilia pathological changes were seen by x-ray in one or both knees in every patient and in the elbows of all but one.[3] The degree of change was proportional to the age and the number of acute episodes.

Bleeding into a single small joint may simulate the clinical and roentgenological features of a sarcoma,[19] chronic hemarthrosis may at times resemble tuberculosis, rheumatoid arthritis, Perthe's disease, syphilis or tumor. History of bleeding from other sites, previous attacks in a young male, slight febrile response, muscular atrophy, and absence of associated features of the above diseases should serve to differentiate.

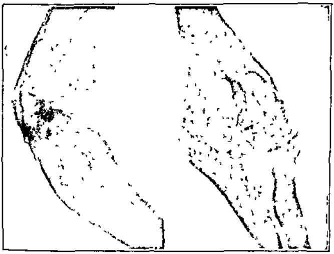


FIG 266 —Hemophilia with organized blood-clot in the capsule of the elbow joint (Holmes and Robbins.)

Treatment.—Known hemophiliacs and males of tainted stock must be protected most carefully. The development of "accident consciousness" on the part of the parents may prevent wounds and abrasions. Violent

tendency as soon as he can comprehend and taught to protect himself. In early adolescence plans for an appropriate vocation should be entertained. Ideally, an afflicted male should be sterilized if he contemplates marriage.

Transfusions of fresh whole blood, plasma, or lyophilized plasma have

can be repeated as often as necessary. Intramuscular and subcutaneous

Symptoms due to local involvement by the disease are briefly noted as follows:—the uveoparotid syndrome consists of a variety of eye symptoms partly due to orbital and partly to neurologic lesions which may cause visual impairment even to the extent of blindness. These are usually preceded by

unless the overlying skin is damaged

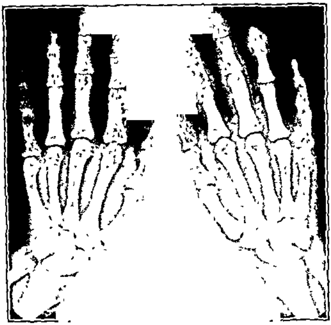


FIG 268 —Roentgenograms of the hands of patient shown in figure 229 showing the characteristic bone lesions of sarcoidosis The fusiform swelling is between rather than at the joints Note narrowing of a few of the joint spaces.

Large hilar masses may cause an irritative cough, but even the massive lung infiltrations do not cause dyspnea, although they may result in chronic right-sided heart strain and polycythemia. Involvement of the myocardium may cause various arrhythmias and even congestive failure. Invasion of the thyroid, pituitary and gonads have been reported to cause myxedema, diabetes insipidus, and eunuchoidism respectively There may be impaired renal function but seldom uremia Peripheral neuropathies, especially involving the cranial nerves may also occur Few diseases in the field of medicine can produce such a varied clinical picture.

Laboratory Findings.—Notable deviations are as follows —high total serum protein due mainly to increased globulins which may equal the albumin. Serum calcium and phosphatase are increased but phosphorus

capsule becomes greatly thickened, and punched-out areas due to subchrondral hemorrhages may appear. Cavitation may develop from intraosseus hemorrhage.[19,29] [illegible]

Bizarre distortions of the joint may supervene. Marginal osteophytes are rare, but fibrous ankylosis begins relatively early.



FIG 265 —Marked contractures of both knees in a severe hemophiliac seven years of age (Key, courtesy of Ann Surg)

The terminal stage leading to hemophilic arthritis may be heralded by subluxations or fibrous and bony ankylosis. In the smaller articulations complete destruction of the joint pattern and even of the bones is not infrequent. Both atrophy and proliferation of bone occur, depending on interference with the nutrition of the bone or excessive flow of blood to the growing epiphyses.[13,35]

X-ray films of an acute hemarthrosis show a bulged out capsule and widened joint space.[1 12 20] Increased density due to the blood is readily apparent Subsequently, opacities in the soft tissues may be visible due to the deposition of iron pigment in the organizing hematomata.[2,14 35 38] Distortion of the joint cleft due to the irregular destruction of cartilage o ether with rarefactions, cysts, and cavities, and eroded subchrondral

Clinical Picture.—It will readily be seen that the clinical manifestations of these lesions will be exceedingly diverse, and often misleading The onset is insidious and the systemic manifestations few, most of the symptoms being caused by mechanical interference due to the infiltration by the characteristic cells There may be malaise, weakness, drowsiness, night sweats, anorexia, mild gastrointestinal discomfort, diarrhea and weight loss

The joint manifestations consist of vague arthralgias in the active phases of the disease and tightness and stiffness due to involvement of the skin of



A/G ratio 0 66

diagnostic. (Fig. 268.)

Active inflammation of the joints has been reported where the process has extended into the synovium from adjacent areas One case is reported in which there was a co-existing polyarthritis indistinguishable from rheumatoid arthritis.[2]

areas appear in more advanced cases.[14,17,33,45] Accelerated epiphyseal

but one[33] The degree of change was proportional to the age and the number of acute episodes.

Bleeding into a single small joint may simulate the clinical and roentgenological features of a sarcoma,[19] chronic hemarthrosis may at times resemble tuberculosis, rheumatoid arthritis, Perthe's disease, syphilis or tumor History of bleeding from other sites, previous attacks in a young male, slight febrile response, muscular atrophy, and absence of associated features of the above diseases should serve to differentiate

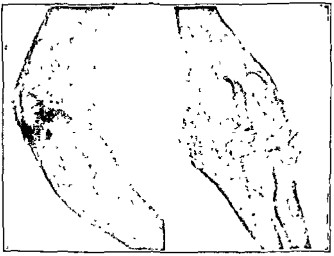


FIG 266.—Hemophilia with organized blood-clot in the capsule of the elbow joint (Holmes and Robbins)

Treatment.—Known hemophiliacs and males of tainted stock must be protected most carefully The development of "accident consciousness" on the part of the parents may prevent wounds and abrasions Violent exertion and play in which trauma is apt to result are best avoided. The child should live as normal a life as possible since overzealous guarding may lead *to chronic invalidism With care and forethought independence and* self reliance may be attained The child should be told of his inherited tendency as soon as he can comprehend and taught to protect himself In early adolescence plans for an appropriate vocation should be entertained Ideally, an afflicted male should be sterilized if he contemplates marriage

Transfusions of fresh whole blood, plasma, or lyophilized plasma have

can be repeated as often as necessary Intramuscular and subcutaneous

advocated as treatment but cannot be recommended. Urethane exerts a suppressive effect, as does *x*-ray therapy. ACTH and cortisone cause prompt involution of lesions and may be of great value when these threaten a vital area. However, their place in long term therapy is not established.[4,6]

BIBLIOGRAPHY

1 Castellanos, A, and Galan, E: Am Jour. Dis Child, **71**, 513, 1946
2 Davis, M. W, and Crotty, R Q Ann Int Med, **36**, 1098, 1952
3 Israel, H. L, and Sones, M Am Pract & Digest Treat, **3**, 18, 1952.
4 Longcope, W T, and Freiman, D G Medicine, **31**, 1, 1952
5 Shay, Harry *et al* Gastroenterology, **19**, 441, 1951.
6 Sones, Maurice *et al* New England Jour Med, **244**, 209, 1951.

capsule becomes greatly thickened, and punched-out areas due to subchrondral hemorrhages may appear. Cavitation may develop from intra-

Bizarre distortions of the joint may supervene. Marginal osteophytes are rare, but fibrous ankylosis begins relatively early.



FIG 265 —Marked contractures of both knees in a severe hemophiliac seven years of age (Key, courtesy of Ann Surg)

The terminal stage leading to hemophilic arthritis may be heralded by subluxations or fibrous and bony ankylosis. In the smaller articulations complete destruction of the joint pattern and even of the bones is not infrequent. Both atrophy and proliferation of bone occur, depending on interference with the nutrition of the bone or excessive flow of blood to the growing epiphyses.[13,35]

X-ray films of an acute hemarthrosis show a bulged out capsule and widened joint space.[1,12,20] Increased density due to the blood is readily apparent. Subsequently, opacities in the soft tissues may be visible due to the deposition of iron pigment in t
Distortion of the joint cleft due to th
ther with rarefactions, cysts, and

normal Cerebrospinal fluid may show increased protein. There may be a

The phalanges, and to a lesser extent the skull and long bones, show rarefactions and punched out areas (Fig. 268) in the medulla without however any periosteal change.

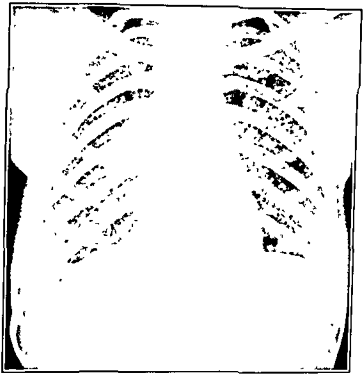


Fig. 269 —Roentgenogram of chest of a twenty-eight year old colored female showing diffuse sarcoidosis of the lungs The diagnosis was proved by lymph-node biopsy. The patient had no complaints and the lesions were discovered during a mass x-ray chest survey The tuberculin test was positive (0 0001 mgm. P.P D)

Diagnosis.—The skin lesions and uveoparotid syndrome may permit diagnosis by inspection The disease should be thought of in any case of chronic lymphadenopathy and splenomegaly. Characteristic x-ray findings

per cent).[6]

Treatment.—The patient should be reassured that he does not have tuberculosis. Evaluation of treatment is difficult because of frequent spontaneous remissions A host of drugs such as arsenic, iodides, gold, Chaulmoogra oil, old tuberculin, calcifirol and nitrogen-mustard have been

areas appear in more advanced cases.[11,17,33,41] Accelerated epiphyseal maturation may be seen early in hemophilia if the child has had repeated hemarthroses.[11,35] In 15 adults with hemophilia pathological changes were seen by *x*-ray *in one or both knees in every patient and in the elbows of all* but one.[35] The degree of change was proportional to the age and the number of acute episodes.

Bleeding into a single small joint may simulate the clinical and roentgenological features of a *sarcoma*,[19] *chronic hemarthrosis may at times* resemble tuberculosis, rheumatoid arthritis, Perthe's disease, syphilis or tumor. History of bleeding from other sites, previous attacks in a young male, slight febrile response, muscular atrophy, and absence of associated features of the above diseases should serve to differentiate.

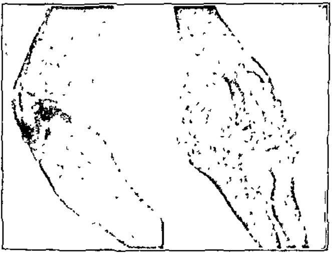


FIG 266.—Hemophilia with organized blood-clot in the capsule of the elbow joint (Holmes and Robbins.)

Treatment.—Known hemophiliacs and males of tainted stock must be protected most carefully. The development of "accident consciousness" on the part of the parents may prevent wounds and abrasions. Violent exertion and play *in which trauma is apt to result are best avoided*. *The* child should live as normal a life as possible since overzealous guarding may lead to chronic invalidism. With care and forethought independence and self reliance may be attained. The child should be told of his inherited tendency *as soon as he can comprehend and taught to protect himself*. In early adolescence plans for an appropriate vocation should be entertained. Ideally, an afflicted male should be sterilized if he contemplates marriage.

Transfusions of fresh whole blood, plasma, or lyophilized plasma have [illegible]

can be repeated *as often as necessary*. *Intramuscular and subcutaneous*

and edema of the overlying tissues. Pain and stiffness in the knee are the

response of articular cavities or of tendon sheaths to any irritant is the exudation of fluid [15] When there is much fluid, the swelling may be hard and may feel like a tumor.[44] About articular ligaments and the tissues binding tendons, trauma is always followed by edema If there is hemorrhage[38] this may subside slowly and remain tender for a long time *When monarticular swellings develop without definite traumatic history or persists, tumor must always be considered in the diagnosis* [22] New growths of any type are not common in these areas [28] Metastatic tumors near the joints, tendon sheaths or bursæ are extremely rare but may cause inflammation Particularly in monarticular arthritis differential diagnosis may be extremely difficult Probably the most common error is that of diagnosing metastatic carcinoma of the spine as osteoarthritis Probably the next most common error is diagnosing a giant cell tumor near a joint as an acute arthritis with effusion All the tumors which arise in bone or in fibrous tissue[41] must be considered [45]

Tumors in these regions can be grouped as follows [50]

Benign

1 Osteochondroma or osteoma
2. Multiple exostoses
3. Chondromas and chondromyxomas
4. Benign giant cell tumors
5 Fibroma
6 Lipoma
7 Hemangioma
8 Osteoid osteoma
9 Eosinophilic granuloma

Malignant

1 Synovioma
2 Fibrosarcoma
3 Liposarcoma
4. Chondromyxosarcoma
5 Osteogenic sarcoma
6 Chondroblastic sarcoma
7 Multiple myeloma
8 Endothelial sarcoma
9. Malignant giant cell tumors
10. Ewing's tumor
11 Metastatic tumors of bones and joints

Lesions simulating tumors

1 Traumatic lesions
2 Infections
3 Osteitis fibrosa cystica
4. Metabolic lesions
5 Villonodular synovitis
6. Cysts of bone in osteoarthritis

Osteochondroma.—These growths arise probably as cartilaginous rests They have a pedicle of bone with a cartilaginous crest They may cause symptoms from pressure if near the joint. They rarely show malignant degeneration. Complete removal usually results in cure They may be single or *multiple.*[42] They may arise from fibrous tissue in any part of the body. In these locations histological examination is necessary for diagnosis The essential points concerning these lesions are as follows:

capsule becomes greatly thickened, and punched-out areas due to subchrondral hemorrhages may appear. Cavitation may develop from intraosseus hemorrhage.[19,29] Villous hypertrophy of the synovial membrane is accompanied by erosion of the cartilage at the joint margins. The articular surface of cartilage may be destroyed in an irregular map-like fashion.[21,22,25] Bizarre distortions of the joint may supervene Marginal osteophytes are rare, but fibrous ankylosis begins relatively early.



FIG 265 —Marked contractures of both knees in a severe hemophiliac seven years of age (Key, courtesy of Ann Surg)

The terminal stage leading to hemophilic arthritis may be heralded by subluxations or fibrous and bony ankylosis. In the smaller articulations complete destruction of the joint pattern and even of the bones is not [illegible]

X-ray films of an acute hemarthrosis show a [illegible] *widened joint space.*[1,12,20] Increased density due to the blood is readily apparent Subsequently, opacities in the soft tissues may be visible due to the deposition of iron pigment in the organizing hematomata.[2,14,35,38] Distortion of the joint cleft due to the irregular destruction of cartilage together with rarefactions, cysts, and cavities, and eroded subchrondral

CHAPTER 51

TUMORS OF JOINTS AND TENDON SHEATHS

By John G Kuhns, M.D. and Theodore A. Potter, M.D.

Many swellings are found in and about the joints and the tendon sheaths. The great majority of these are not new growths. Particularly in chronic arthritis thickening and swelling are seen within and about the joints, in the tendon sheaths and on the flexor surfaces of the muscles. The proliferation of the synovial membrane in rheumatoid arthritis may simulate tumor formation Conversely, fibrosarcoma,[22] especially that arising from the synovial membrane, gives at first the appearance of early rheumatoid arthritis. Tumors arising in bone both benign and malignant lead to swelling

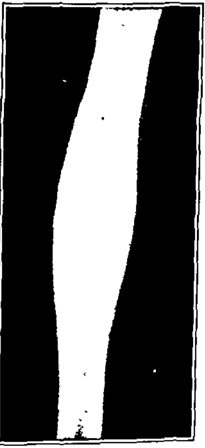


Fig 270 —Ewing's tumor of femur, showing typical x-ray appearance—ovoid femoral shaft

dred of these osteocartilaginous bodies, some loose, some attached to
gradually but
The symptoms
he elbow joints
are the ones most commonly involved. Treatment is complete removal of

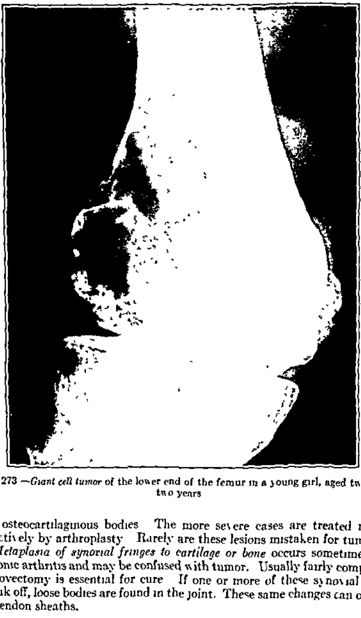


FIG 273 —*Giant cell tumor* of the lower end of the femur in a young girl, aged twenty-two years

the osteocartilaginous bodies The more severe cases are treated most effectively by arthroplasty Rarely are these lesions mistaken for tumors

Metaplasia of synovial fringes to cartilage or bone occurs sometimes in chronic arthritis and may be confused with tumor. Usually fairly complete synovectomy is essential for cure If one or more of these synovial villi break off, loose bodies are found in the joint. These same changes can occur in tendon sheaths.

mesenchymal

Osteochondroma.—Usually

involved tendon. If these lesions increase in size rapidly, the

malignant change must not be forgotten If they are causing symptoms they should be excised.[31]

discussed in another section It probably represents

r of tendon sheaths.



FIG 281.—*Cellular synovioma or low grade fibrosarcoma* of a knee with invasion of the bursal and joint cavities, showing lobulated character of the growth (Geschicter, courtesy of Am Jour Cancer)

reported at th and growth is

ey are

and or

movable. They are

encapsulated n; metastases do not

occur. Microscopically, they are composed chiefly of fibroblasts with a scattering of xanthoma and giant cells, blood pigment and cholesterin

collected

They are most common between the ages of twenty and fifty, especially in males.

The order of frequency of joint involvement is: knee, elbow, ankle, hip and shoulder, and occasionally in the hands and feet.[1]

Osteochondromatosis is usually a monarticular disease. This may be either inflammatory, traumatic or both. Many loose bodies develop or are found in the joint cavity. In arthritis at operation one often finds fifty to a hun-



FIG 271.—*A*. Osteoarthritis of the cervical spine *B* The same spine four months later with metastatic carcinoma and collapse of fourth cervical vertebra

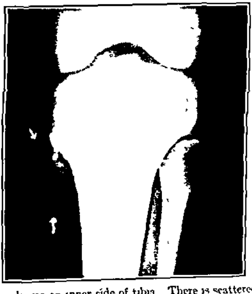


FIG 272.—*Osteochondroma on inner side of tibia* There is scattered ossification with the tumor. There was pain on motion and slight swelling of the knee joint

Malignant Tumors of Bone Causing Articular Symptoms.—All slowly growing malignant tumors whether of bone or of soft tissues if near a joint will give the same symptoms as a slowly developing arthritis—swelling, tenderness and disability. This has been observed within the knee joint where slight pain and stiffness of two weeks' duration led to the finding of a small osteolytic sarcoma in the lower end of the femur. Pain and flexion deformity at the hip originally diagnosed as arthritis was found to be lymphosarcoma at operation. Such instances can be duplicated in any arthritic clinic



FIG 280—Chondrosarcoma of upper end of humerus causing articular symptoms in shoulder. Note dense irregular calcification within the humeral head

Tumors of Tendon Sheaths.—The following tumors have been reported:[28]

Benign:[29]

a. Chondroma and osteochondroma
b. Ganglion
c. Giant cell tumor
d. Uncommon: fibroma, lipoma, hemangioma, lymphangioma

Malignant.

a Fibrosarcoma (rare)
b. Chondrosarcoma.

Primary tumors of tendons are very rare, the most common being sarcoma.[57] Primary neoplasms of tendon sheaths are more frequent.[4] The

majority of cases being in the third to sixth decades. These workers thought that a mild, constant and unnoticeable trauma and possibly infection were important secondary factors in the production of the tumors. The primary factor was an alteration in lipoid metabolism.

When xanthomas involve joints,[66] pain and heat may or may not be present, examination of the aspirated fluid reveals an elevated cholesterol content. The tumor does not metastasize unless sarcomatous degeneration occurs (rare). It is prone to recur locally if all tumor cells are not destroyed at its removal.



Fig 275 —Giant cell tumor of the upper end of the left humerus in a man, aged thirty-four years

Fibroma.—This is a tumor composed chiefly of connective tissue. Some

may involve bones,
ressure or from dis-
ired. Trauma may
cause more rapid growth and probably sarcomatous change

Lipoma.[20]—This tumor may arise from extracapsular fat or occasionally may be intracapsular (lipoma arborescens, see Fig 285)

tendon sheaths. myeloma, xanthoma, granulation tissue tumor and xanthomatous giant cell tumor It is seen most often in the tendon sheaths of the hands and feet, but has been



FIG. 281 —Cellular synovioma or low grade fibrosarcoma of a knee with invasion of the bursal and joint cavities, showing lobulated character of the growth (Geschicter, courtesy of Am Jour Cancer)

reported at the wrists and ankles[77] The onset is insidious and growth is usually slow Although such tumors may attain the size of a fist, they are rarely larger than a walnut.[78] On examination, they appear as round or lobulated smooth masses over which the skin is freely movable. They are encapsulated and are easily shelled out at operation, metastases do not occur Microscopically, they are composed chiefly of fibroblasts with a scattering of xanthoma and giant cells, blood pigment and cholesterin crystals.

Bursal Tumors.[61]—Until 1934, only 30 tumors of bursæ could be collected from the literature. The prepatellar bursa was affected most often Such tumors include chondroma, osteochondroma, myxoma, and fibrosarcoma

Loose bodies may be partly calcified or ossified. They are usually readily detected by x-ray.

arise from the cortex of the bone near the epiphyseal line. Bursae may develop over them. Removal is indicated when they cause deformity or pain.

Chondroma and Chondromyxomas.—The gradations of these tumors into a single type of tissue, osteochondroma in which bone predominates,[42] benign chondroma in which cartilage predominates and chondromyxoma with myxoma and early cartilage formation indicate a fairly close relationship between them.[55] In chondroma and chondromyxoma complete excision and cauterization are required for cure.[39]

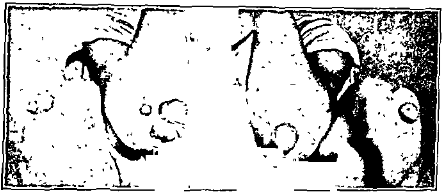


FIG 274 —Multiple xanthomata near joints (knees and elbows) in young colored male

Xanthomatous Giant Cell Tumors.—Histologically these are composed of foam cells, loose connective tissue and giant cells. This is the second commonest form of benign joint tumor, very rarely diagnosed preoperatively.[35] The usual location is the knee joint.[3] It may occur at any age and grows slowly. The symptoms may simulate those of rupture of the medial meniscus or traumatic hydrarthrosis.[49]

Points suggesting a diagnosis of xanthoma of the knee joint: obscure intermittent joint swelling, pain, free fluid in the joint, occasional locking of the joint and a movable tumor (most often medial to the patella). Dark or sanguinous fluid may be found on aspiration of the joint; this may contain a large amount of cholesterol.

Galloway, Broders and Ghormley[29] reported a clinical and pathologic study of xanthoma of tendon sheaths and synovial membranes. They showed that these were slowly growing painless tumors averaging approximately five years before their discovery. The new growths vary in shape and size, but average 2 5 by 2 cm when first seen by the physician. In ... firm round or oval lesions, occurring most often ... common on the upper extremity as on ... patients was forty-four years, with the

Malignant Tumors of Bone Causing Articular Symptoms.—All slowly growing malignant tumors whether of bone or of soft tissues if near a joint will give the same symptoms as a slowly developing arthritis—swelling, tenderness and disability. This has been observed within the knee joint where slight pain and stiffness of two weeks' duration led to the finding of a small osteolytic sarcoma in the lower end of the femur. Pain and flexion deformity at the hip originally diagnosed as arthritis was found to be lymphosarcoma at operation. Such instances can be duplicated in any arthritic clinic

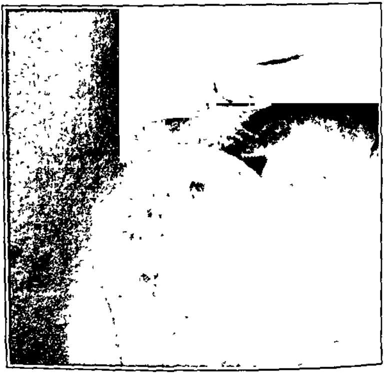


FIG 280 —Chondrosarcoma of upper end of humerus causing articular symptoms in shoulder Note dense irregular calcification within the humeral head

Tumors of Tendon Sheaths.—The following tumors have been reported:

Benign:[23]

a. Chondroma and osteochondroma
b Ganglion
c. Giant cell tumor
d. Uncommon· fibroma, lipoma, hemangioma, lymphangioma

Malignant:

a Fibrosarcoma (rare)
b. Chondrosarcoma.

Primary tumors of tendons are very rare, the most common being sarcoma[57] Primary neoplasms of tendon sheaths are more frequent.[4] The

The possibility of disseminated hemangiomas of bone must be borne in mind when diffuse osteolytic lesions of the skeletal system are seen [36]

There are a number of lesions more common in youth than in adults which appear at times near the ends of the long bones These lesions called granuloma may be new growths or the reaction to a mild infection or noxious agent. One of these, eosinophilic granuloma, often presents

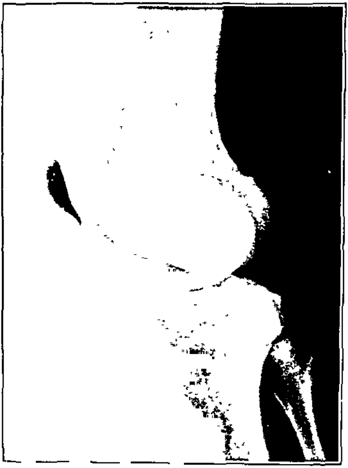


Fig 277 —Osteogenic sarcoma of lower end of the femur showing "sun-ray like" projections of condyle anteriorly

multiple bony lesions These are wholly osteolytic lesions Symptoms may not be present but usually there are pain, swelling and dysfunction which may simulate a slowly developing arthritis. Spontaneous resorption of the lesion has been observed The usual treatment is curettage of the lesion or x-ray treatment

basic cell of such tumors is the synovial cell.[58] The latter is of mesenchymal origin and possesses multipotential qualities in that it may become cartilage or bone or may revert to fibroblasts.[59]

Osteochondroma.—Usually causes only mild pain on motion of the involved tendon If these lesions increase in size rapidly, the possibility of ... If they are causing symptoms

... section. It probably represents

thoma, granulation tissue tumor and xanthomatous giant cell tumor. It is seen most often in the tendon sheaths of the hands and feet, but has been



FIG 281 —Cellular *synovioma* or low grade fibrosarcoma of a knee with invasion of the bursal and joint cavities, showing lobulated character of the growth (Geschicter, courtesy of Am Jour Cancer)

reported at the wrists and ankles.[12] The onset is insidious and growth is usually slow. Although such tumors may attain the size of a fist, they are rarely larger than a walnut[77] On examination, they appear as round or *lobulated smooth masses over which the skin is freely movable* They are encapsulated and are easily shelled out at operation, metastases do not occur Microscopically, they are composed chiefly of fibroblasts with a scattering of xanthoma and giant cells, blood pigment and cholesterin crystals.

Bursal Tumors.[61]—Until 1934, only 30 tumors of bursæ could be collected from the literature. The prepatellar bursa was affected most often. Such tumors include chondroma, osteochondroma, myxoma, and fibrosarcoma.

Large extracapsular lipomas may be diagnosed by roentgen-ray [66] Intra-articular limpomas (lipoma arborescens) represent hypertrophied synovial tissues distended with fat. They are very rare. They may interfere with function when expanding between muscles [69]

Treatment, when necessary, is surgical removal.[70]

Hemangioma.—These are extremely rare.[42] Trauma is not a factor in

have been found in over 10 per cent of a large series of routine autopsies; these rarely result in compression of the cord, but if they occur in young males, they seem to produce symptoms rather often. Although the hemangioma may show no demonstrable roentgen signs, there is usually

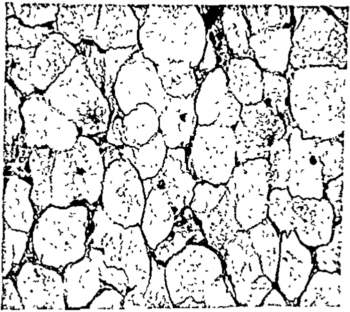


Fig. 276—*Photomicrograph of a xantholipoma of the knee joint.* (Geschicter, courtesy of Am Jour Cancer.)

seen a reduction of bone density between parallel trabeculæ which are *increased in density*.[46] If the diagnosis is made before the cord has been irreparably damaged, roentgen therapy may effect a cure. Laminectomy is advised when paraplegia has developed [72]

Primary hemangioma of the vertebra is comparatively common but rarely gives rise to symptoms [9] The diagnosis can usually be made by the coarse meshed appearance of the vertebra from vertical striations alternating with large areas of bony rarefaction If the lesion produces neurologic symptoms, surgery should be attempted if the patient is a good operative

Malignant Tumors of Bone Causing Articular Symptoms.—All slowly growing malignant tumors whether of bone or of soft tissues if near a joint will give the same symptoms as a slowly developing *arthritis*—swelling, tenderness and disability. This has been observed within the knee joint where slight pain and stiffness of two weeks' duration led to the finding of a small osteolytic sarcoma in the lower end of the femur. Pain and flexion deformity at the hip originally diagnosed as arthritis was found to be lymphosarcoma at operation. Such instances can be duplicated in any arthritic clinic.



FIG. 280.—Chondrosarcoma of upper end of humerus causing articular symptoms in shoulder. Note dense irregular calcification within the humeral head.

Tumors of Tendon Sheaths.—The following tumors have been reported:[?]

Benign:[25]

a. Chondroma and osteochondroma
b. Ganglion
c. Giant cell tumor
d. Uncommon: fibroma, lipoma, hemangioma, lymphangioma

Malignant:

a. Fibrosarcoma (rare)
b. Chondrosarcoma.

Primary tumors of tendons are very rare, the most common being sarcoma.[57] Primary neoplasms of tendon sheaths are more frequent.[4] The

tion to their presence.[53] They vary greatly in their rate of growth and invasiveness. Most of them can be treated by radical excision and roentgen therapy.[31] This is especially true with fibrosarcoma.[24]



FIG 278 —*Synovial sarcoma* of the elbow (Knox, courtesy of Am. Jour Cancer)



FIG. 279.—Multiple myeloma, producing articular symptoms at hip and spine Note mottled areas of bone destruction in upper femur and innominate bone.

Osteochondroma.—Usually causes only mild pain on motion of the involved tendon. If these lesions increase in size rapidly, the possibility of malignant change must not be forgotten. If they are causing symptoms they should be excised.[81]

Ganglion will be discussed in another section. It probably represents degenerated connective tissue[17] or herniated synovium.

The giant cell tumor is probably the commonest tumor of tendon sheaths. It has also been called an endothelioma, myeloma, giant myeloma, xanthoma, granulation tissue tumor and xanthomatous giant cell tumor It is seen most often in the tendon sheaths of the hands and feet, but has been



FIG 281 —Cellular *synovioma* or low grade fibrosarcoma of a knee with invasion of the bursal and joint cavities, showing lobulated character of the growth (Geschicter, courtesy of Am Jour Cancer)

reported at the [illegible] usually slow [illegible] rarely larger th[illegible] lobulated smoo[illegible] [illegible]ire encapsulated and are easily [illegible] [illegible]not occur Microscopically, they [illegible] a scattering of xanthoma and giant cells, blood pigment and cholesterin crystals.

Bursal Tumors[81]—Until 1934, only 30 tumors of bursæ could be collected from the literature The prepatellar bursa was affected most often Such tumors include chondroma, osteochondroma, myxoma, and fibrosarcoma

Osteoid osteoma[33] has given the appearance of arthritis in the writers' experience. After x-ray not uncommonly it is considered a malignant tumor until pathologic study. This is a *small oval or rounded area* in the bone with peripheral bony sclerosis and a less dense central area It is usually single and more common in males. Pain is mild. There may be swelling, tenderness and disability. Surgical removal is followed by cure

Malignant Synovioma (Fibrosarcoma or Synovial Sarcoma).—*Synoviomata, or tumors of articular synovial membrane, are infrequent.* The diagnosis is seldom made clinically.[2] The prognosis is usually unfavorable because of local recurrence and metastases.[21]

King described 7 cases of synovial sarcomata originally attached to a tendon sheath. In 6 of the 7, excision was followed by recurrences or metastases. Wide excision together with radium implantation gave the only one satisfactory result.[11]

The tumor may at times be cystic and may show a bluish discoloration over its surface. It is often very soft. Microscopically it is composed of *two types of cells—one, cuboidal (epithelium or endothelium) and a smaller* spindle shaped cell.[37]

Characteristics of Synovioma

Origin. synovial membrane.

Age incidence: any age, usually affecting young persons

Order of incidence of joint involvement: knee, finger, ankle, metatarsus.

May be of low or high grade malignancy,[25] they are usually highly malignant[6]

Symptoms: if the knee is involved—dull aching pain, made worse on weight bearing; may exist without impairment of joint function. Spontaneous hemarthrosis may occur.[25]

[illegible]

Metastasis: may occur to the lungs.

Treatment: radical wide excision, often amputation Tumors are not *radiosensitive*[27]

Prognosis: poor; 20 per cent five-year survivals—*no survivals over ten* years; tendency to recur following excision[19]

Other primary malignant tumors of the joints or tendon sheaths are quite rare.[7] Tumors both primary and metastatic occasionally will invade tendons and articular cavities from surrounding areas.[18] Those from the bones are usually osteogenic sarcomas or chondromyxosarcoma rarely the malignant giant cell tumor which has broken through the bone into the knee joint.[37]

[illegible] *multiple* [illegible] adjacent [illegible] ile they may cause symptoms by invasion, pathological fracture [illegible] ls atten-

Malignant Tumors of Bone Causing Articular Symptoms.—All slowly growing malignant tumors whether of bone or of soft tissues if near a joint [illegible] leveloping arthritis—swelling, [illegible] bserved within the knee joint [illegible] duration led to the finding of a [illegible] the femur. Pain and flexion deformity at the hip originally diagnosed as arthritis was found to be lymphosarcoma at operation. Such instances can be duplicated in any arthritic clinic.



Fig. 280.—Chondrosarcoma of upper end of humerus causing articular symptoms in shoulder. Note dense irregular calcification within the humeral head.

Tumors of Tendon Sheaths.—The following tumors have been reported:[20]

Benign:[21]

a. Chondroma and osteochondroma
b. Ganglion
c. Giant cell tumor
d. Uncommon fibroma, lipoma, hemangioma, lymphangioma.

Malignant:

a. Fibrosarcoma (rare)
b. Chondrosarcoma.

Primary tumors of tendons are very rare, the most common being sarcoma.[57] Primary neoplasms of tendon sheaths are more frequent.[4] The

basic cell of such tumors is the synovial cell[58] The latter is of mesenchymal origin and possesses multipotential qualities in that it may become cartilage or bone or may revert to fibroblasts[45]

Osteochondroma —Usually causes only mild pain on motion of the involved tendon If these lesions increase in size rapidly, the possibility of malignant change must not be forgotten If they are causing symptoms they should be excised[61]

Ganglion will be discussed in another section. It probably represents

thoma, granulation tissue tumor and xanthomatous giant cell tumor. It is seen most often in the tendon sheaths of the hands and feet, but has been



FIG 281 —Cellular *synovioma* or low grade fibrosarcoma of a knee with invasion of the bursal and joint cavities, showing lobulated character of the growth (Geschicter, courtesy of Am Jour Cancer)

reported at th
usually slow
rarely larger t
lobulated smoc
encapsulated and are easily shelled out at operation; metastases do not occur. Microscopically, they are composed chiefly of fibroblasts with a scattering of xanthoma and giant cells, blood pigment and cholesterin crystals.

Bursal Tumors.[61]—Until 1934, only 30 tumors of bursæ could be collected from the literature The prepatellar bursa was affected most often Such tumors include chondroma, osteochondroma, myxoma, and fibrosarcoma.

... cell.[58] The latter is of mesenchymal

Osteochondroma.—Usually causes only mild pain on motion of the involved tendon If these lesions increase in size rapidly, the possibility of ... If they are causing symptoms ...

... section It probably represents ...

thoma, granulation tissue tumor and xanthomatous giant cell tumor. It is seen most often in the tendon sheaths of the hands and feet, but has been



FIG 281 —Cellular *synovioma* or low grade fibrosarcoma of a knee with invasion of the bursal and joint cavities, showing lobulated character of the growth (Geschicter, courtesy of Am Jour Cancer)

reported at the wrists and ankles [12] The onset is insidious and growth is usually slow Although such tumors may attain the size of a fist, they are rarely larger than a walnut [71] On examination, they appear as round or lobulated smooth masses over which the skin is freely movable They are encapsulated and are easily shelled out at operation, metastases do not occur. Microscopically, they are composed chiefly of fibroblasts with a scattering of xanthoma and giant cells, blood pigment and cholesterin crystals.

Bursal Tumors.[81]—Until 1934, only 30 tumors of bursæ could be collected from the literature The prepatellar bursa was affected most often Such tumors include chondroma, osteochondroma, myxoma, and fibrosarcoma.

Malignant Tumors of Bone Causing Articular Symptoms.—All slowly growing malignant tumors whether of bone or of soft tissues if near a joint will give the same symptoms as a slowly developing arthritis—swelling, tenderness and disability This has been observed within the knee joint where slight pain and stiffness of two weeks' duration led to the finding of a small osteolytic sarcoma in the lower end of the femur. Pain and flexion deformity at the hip originally diagnosed as arthritis was found to be lymphosarcoma at operation. Such instances can be duplicated in any arthritic clinic.



Fig. 280.—Chondrosarcoma of upper end of humerus causing articular symptoms in shoulder Note dense irregular calcification within the humeral head

Tumors of Tendon Sheaths.—The following tumors have been reported.[20]

Benign:[28]

a. Chondroma and osteochondroma
b Ganglion
c. Giant cell tumor
d. Uncommon: fibroma, lipoma, hemangioma, lymphangioma.

Malignant:

a. Fibrosarcoma (rare)
b. Chondrosarcoma.

Primary tumors of tendons are very rare, the most common being sarcoma.[57] Primary neoplasms of tendon sheaths are more frequent.[4] The



FIG 282 —Fleshy tumor of the palm and base of the index finger present since childhood and a localized manifestation of a generalized von Recklinghausen's *neurofibromatosis;* no functional impairment Photograph of the excised specimen shows plexiform neurofibroma with one myxomatous portion (the white mass attached to the lower margin). (Brunschwig, courtesy of Surgery.)

Glomus Tumors.—These are rather rare tumors most commonly found in or about the fingers or toes. They are usually small, subcutaneous nodules, blue or purple in color. In their development they become quite painful Essentially they consist of a neuromyoarterial nodule which regulates blood flow and maintains the temperature of the extremity.



FIG. 283 —Coarsely nodular tumor of two years duration in a forty-four year old woman Soft mass easily shelled out arose from flexor tendon sheath Microscopic diagnosis *xanthoma* (Brunschwig, courtesy of Surgery)



FIG 284 —Anteroposterior and lateral views of knee joint, the shadow of a *lipoma* may be seen beneath and mesial to the quadriceps tendon. (Ghormley, courtesy of Minnesota Med.)

mits muscular exercise which causes an increase in local circulation and enough mechanical pressure to force the accumulated fluid into channels for its removal The gentle massage produced by motion promotes drainage and decreases the effusion

Following injection of local anesthesia, the joint should be strapped or wrapped snugly with an elastic bandage. The strapping is changed in four or five days when the swelling decreases. Strapping or the application of an elastic support is maintained for about two weeks after clinical signs disappear.

[illegible]

The technic is described in Chapter 18

Some authors recommend **Ethyl chloride sprays**,[27,30] others prefer **procaine iontophoresis** over procaine injections[94] or ethyl chloride spray.[69] The technic of procaine injection is as follows:[63,97]

1 The subject should be given a barbiturate thirty minutes before injection.
2. The area to be injected should be washed with soap and water, followed by the application of an antiseptic solution
3 One or 2 per cent procaine is injected directly into the most tender subcutaneous tissues, withdrawing the barrel of the syringe before injection to prevent entering the general circulation If possible, the tissue should be injected until no tender or painful areas remain on palpation or on ankle or foot motion.
4. [illegible]
5. [illegible] made at any one time (to reduce possible avenues of entrance of infection).
6. From 10 to 30 cc. of solution should be injected
7. The injection may be repeated in twenty-four hours if pain is still present

Contraindications to Procaine Injections in Sprains (1) An infected or potentially infected area at or near the site of sprain (2) An open wound (3) Idiosyncrasy to procaine; another local anesthetic may then be selected. (4) Rupture of a medium sized or large vessel at or near the area of sprain.

Management Following Procaine Injection.—After injection an elastic bandage is wrapped snugly around the joint. In ankle sprains the foot and ankle then are used normally except for strenuous activity (running, jumping, etc.). The individual is instructed to move the foot, even while sitting, and not keep it immobile in one position even for short periods The elastic bandage may be removed by the patient and the part rewrapped in an hour or two to prevent possible circulatory constriction

By such management, McMaster[70] obtained excellent results in "over 200 cases." He stated that many had no disability; in others, disability rarely lasted longer than twenty-four or forty-eight hours. Reinjection was never necessary; often motion of the ankle was normal or nearly so on the day following injection. For several days, forced passive motions as

Simple excision leads to cure. This is a tumor which can be overlooked in arthritis since the extremities usually show some pain.

Diseases Simulating Tumors.—There are a number of widely dissimilar pathological lesions which can give a clinical picture which may be confused with tumors.[57] The commonest of these are trauma, infections,[45] osteitis fibrosa cystica and metabolic disturbances such as rickets and scurvy.

about joints and tendon sheaths is vitamin D intoxication. Here swelling and deposition of calcium are often found about or in the capsule of joints Discontinuance of the large doses of vitamin D causes a decrease in the swelling and a gradual removal of the deposits of calcium.



FIG 285 —*Arborescent lipoma* from knee joint. (Geschicter, courtesy of Am. Jour. Cancer.)

tumor fail to resemble arthritis after long observation. In tumors there is a steady growth, and not periods of exacerbation and regression. Pain is a more common symptom particularly on motion in arthritis. In benign tumors pain is rare and is commonly found only in the later stages of

injection procedure depended upon two factors.

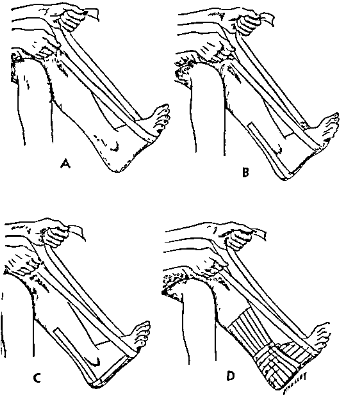


FIG 289 —Adhesive strapping of the ankle (especially useful for sprains, and acute pain of foot strain) Adhesive strips are 1 inch wide Place a pad of thin felt, flannel or gauze over the tendo Achilles before strapping (Dickson and Diveley, Functional Disorders of the Foot, courtesy of J. B Lippincott Company)

1. Complete adequate initial intraligamentous injection to produce total elimination of pain and tenderness; and
2. Continued use and motion of the foot and ankle; this second point he considered the more important.

Stiffness, swelling and pain result and persist if the injection fails to relieve pain, causing the patient to limp and fail to use the part normally, or if he is apprehensive and does not move the part as instructed.

the life of the patient ... tumors wh ... elevated on ... the prostate gland The location and character of osseous changes can often yield a diagnosis through x-ray examination to the experienced roentgenologist Osteolysis, osteosclerosis or combinations of the two can give relatively characteristic pictures at various age periods. However ... the physician must ... *ut joints and tendons*

BIBLIOGRAPHY

1 AHLBERG, A Acta chir. Scandinav, 89, 75, 1943.
2 AITKEN, A. P Jour Bone and Joint Surg, 23, 950, 1941
3 ANDERSON, S R Acta radiol, 26, 11, 1945
4 AREY, J B Jour Pediat, 35, 778, 1949
5 BEDRICK, J J, and ZAWADZKI, S A Mil Surg, 97 ...
6 BENNETT, G F ...
7 BENNETT G ...
8 BLACK, W C ...
9 BLACKFORD, L ...
10 BLUM, S D ... 1947
11 BRIGGS, C D Ann Surg., 115, 413, 1942
12. BRUNSCHWIG, A Surgery, 5, 101, 1939.
13 CHANDLER, F A., and KOELL, H I Arch Surg, 60, 294, 1950.
14 CHAROCHE, H Arch Surg, 44, 1038, 1942
15 CHERRY, J H, and GHORMLEY, R K Am Jour Surg, 52, 319, 1941
16 COBEY, M C Arch Surg., 46, 465, 1943.
17 COLEY, W B, and HIGINBOTHAM, N L. Ann. Surg, 128, 533, 1948
18 COLEY, W B, and PIERSON, J C. Surg, 1, 113, 1937
19. CRISTOL, D S, and GILL, A B Jour Am Med Assn, 122, 1013, 1943
20 DARGEON, H W Jour Pediat, 30, 716, 1947.
21 DESANTO, D A, TENNANT, R., and ROSAHN, P D Surg Gynec and Obst, 72 951, 941.
22 DESANTO, D A, and WILSON, P D Jour Bone and Joint Surg, 21, 531, 1939
23 DOYLE, R W. Practitioner, 156, 267, 1946
24 EVELETH, M S ... 16, 27, 1943
25 FERGU ... 1942
26 FISHE ...
27 FOOT, ... Med Sci, 215, 479, 1948
28 FOSTER, L N. Am. Jour Path, 23, 567, 1947
29 GALLOWAY, J D B, BRODERS, A C, and GHORMLEY, R K Arch Surg, 40, 485, 1940
30 GESCHICKTER, C F, and LEWIS, D Am Jour of Cancer, 22, 96, 1934
31 GHORMLEY, R K Minn Med, 17, 62, 1934
32 GHORMLEY, R K, and DOCKERTY, M B Jour Bone and Joint Surg, 25, 30, 1943
33 HAGGART, G E Jour Bone and Joint Surg, 24, 438, 1942
34 HAGGART, G E, and HARE, H F Ann. Surg, 124, 228, 1946.
35 HARMON, P H Arch Surg, 47, 359, 1943.
36 HARTZ, P H Arch Path, 40, 88, 1945
37 HATCHER, C H Ann Surg, 122, 1016, 1945
38 ILMER, A E, MEILSTRAP, D B, and BOGART, F B Radiology, 46, 597, 1946.
39 JAFFE, H L., and LICHTENSTEIN, L. Bull. Hosp Joint Dis, 2, 3, 1941
40. JANSEN, K F Acta Radiol, 24, 385, 1943
41 KAPLAN, I I Am Jour Dis Child 65, 785, 1943
42 KANTZ, F G Radiology, 45, 162, 1945.
43 KEIM, H. L Clinics, 3, 866, 1944.
44 KELLY, LEM C. New York State Jour Med, 40, 1607, 1940

twelve hours. He applied gentle massage and light manipulation following the injections

Principles of Adhesive Strapping.—1 Strapping must not interfere with the circulation of blood.

2. Straps must be of proper width and length varying with the part strapped.

Adhesive Strapping of the Sprained Ankle.—As a preliminary measure, the

Before applying the adhesive the patient holds tightly both ends of a short piece of muslin bandage which passes under his forefoot and maintains flexion of the foot The pull on the forefoot is not relaxed until the adhesive bandage is completed

1 Hold the foot relaxed at slightly less than a right angle
2 For an average sized adult, prepare 7 strips of adhesive 20 inches long $\frac{3}{4}$ to 1 inch wide, and 7 strips varying from 8 to 15 inches long and 1 inch wide
3 Begin with a long strip about an inch above and posterior to the internal malleolus and carry this under the sole of the foot and up on the outer side terminating 5 or 6 inches above the external malleolus Next apply a short strip with its midportion low over the tendo Achilles (which has been covered with gauze) and extend each end

4

A roller bandage is applied over the strapping and may be removed after the adhesive has become firmly attached to the skin

Following recovery from severe sprains one may wear a leather or canvas anklet with whalebone or thin steel springs as reinforcement, areas are cut out at the malleoli and heel to prevent undue pressure This usually extends for 3 or 4 inches above the ankle, and laces anteriorly In some individuals, it may be advisable to wear such a support for eight to twelve months after a severe sprain. One also may temporarily raise the heel on the affected side about $\frac{1}{2}$ inch if required.

Adhesive Strapping of a Sprained Wrist.—Place a hard roller bandage 3 inches wide and $\frac{1}{2}$ inch thick on the more painful side of the wrist

Fasten this in place by $\frac{1}{2}$ inch strips of adhesive, several layers of adhesive may be applied over this

Strapping of the Sprained Knee.[31]—The knee is a relatively weak structure exposed to trauma and is often injured Prolonged disability may result from strains which produce rotation and lateral motion

A sprain or tear of the lateral ligaments (usually the internal) is the commonest injury to the knee.—Displacement of the semilunar cartilage is frequently *associated with tear of a lateral ligament (in some cases producing no locking).* The cruciate ligaments (especially the anterior) often are

45. KRAUSE, G R: Am. Jour. Roentgenol., 52, 620, 1944
46. KUHNS, J G: Arch. Surg, 46, 687, 1943
47. LACKMAN, E: Surg, Gynec and Obst, 85, 767, 1947.
48. LEWIS, R W: Am Jour. Roentgenol., 44, 170, 1940.
49. MACCARTY, W C.. Jour. Inter. Coll. Surg, 10, 263, 1947.
50. MEYERDING, H W.: Jour. Bone & Joint Surg, 27, 1, 1945
51. MEYERDING, H. W, and VARNEY, J. H. Minn. Med, 30, 159, 1947.
52. MURRAY, M R, STOUT, A P, and POGOGEFF, I. A: Ann. Surg, 120, 843, 1944
53. O'DONOGHUE, D H: Jour Bone and Joint Surg, 24, 940, 1942
54. PIERCE, C B, and EAGLESHAM, D C Radiology, 39, 655, 1942
55. REGAN, J M, BICKEL, W. H, and BRODERS, A C: West. Jour Surg, 54, 87, 1946
56. ROBILLARD, G O: Am Jour Surg, 51, 442, 1941.
57. SACHS, A E, and RUBINSTEIN, L Surgery, 20, 385, 1946
58. SCHUMANN, G, and FINSTERBUSCH, R.: Arch. f klin Chir, 202, 269, 1941.
59. SERVICE, A C. Clinics, 4, 63, 1945
60. SHEPHERD, J A Brit Jour Surg, 30, 179, 1942
61. SHELLITO, J. G: Surg, Gynec and Obst, 86, 465, 1948
62. SHERMAN, M S Jour Bone and Joint Surg, 29, 483, 1947.
63. SHORWAN, L M Brit. Jour. Radiol, 19, 474, 1946
64. SIGURDSON, L A Canadian Med Assn Jour., 53, 274, 1945
65. SNYDER, C H: Am. Jour Surg, 55, 67, 1942
66. SPITZ, S Jour Am Med Women's Assn., 6, 209, 1951.
67. STEPHENS, V R. Arch Surg, 49, 9, 1944
68. SUTHERLAND, C G Radiology, 3, 420 1942
69. WILSON, D A Surg, Gynec. and Obst., 80, 550, 1945
70. WAHLGREN, F Acta Path. et Microbiol Scandav., supp. 26, 77, 1936.
71. WHITE, J R Surg Gynec and Obst, 38, 489, 1924.
72. WOOD, C. Kentucky Med Jour, 41, 82, 1943
73. ZADEK, I Bull Hosp. Joint Dis, 6, 29, 1945.

injured; other tendons and ligaments surrounding the joint are rarely injured.

A double or bipartite patella may appear as a joint body. Such a patella rarely causes difficulty unless the knee is injured. The extra patella may be removed if necessary.

A twisting strain in older individuals may produce loose bodies in the knee joint with resulting pain and swelling of the joint. Most of these loose bodies come from fragmentation of an osteochondritis. Loose fragments (with an osseous center) may grow in the joint and cause locking of the joint. They should be removed as soon as they are discovered.

1. Strips of adhesive 1 inch wide and about 3 inches less than the circumference of the limb are used.
2. The first strip is placed transversely over the middle of the patella.
3. Other strips are placed above and below, overlapping the preceding ones.
4. The second layer of strapping is applied vertically, beginning over the patella and coming within about ½ inch of the upper and lower borders of the preceding strapping; further strips are placed on either side of this.
5. A third layer of strapping is applied horizontally exactly as the first layer, except that strips are somewhat longer and come within an inch or two of encircling the leg.
6. A roller bandage may be applied over this for several hours.

to aid the lymphatics in removing the serous effusion. Favorable results were obtained with static current in sprains and in acute injuries of joints or muscles.

O'Donnell outlined the following program to restore an injured knee to functional activity:[82]

1. Application of cold compresses to the joint for a day or two if much effusion is present.
2. Heat to the upper portion of the thigh, followed about the second or third day by gentle massage of the upper third of the thigh, stroking upward.
3. Muscle setting exercises of the quadriceps to prevent atrophy of this muscle.
4. ... heat to the knee
5. ... teral and rotatory movements must be avoided. Between these ...ents, the knee is immobilized by a posterior molded plaster splint or by adhesive ... additional protection by felt pads placed laterally.
6. ... prevented by early restoration ... crutches.

PART X

REGIONAL DISORDERS OF JOINTS AND RELATED STRUCTURES

CHAPTER 52

TRAUMATIC ARTHRITIS AND ALLIED CONDITIONS

By Nathan R. Abrams, M.D

The term "*traumatic arthritis*" should be discarded, but if used at all, should be reserved for joint lesions produced by acute trauma. In the past, chronic joint disease produced by minor, repeated trauma or "micro-trauma" (the result of minute force applied internally or externally so that no lesion such as hemorrhage, exudation, destruction of cells or neurosis is immediately evident)[34] or from trauma incident to poor posture, athletic activities, occupation, etc ,[12] have been included in this term However, since the *end result* in the latter *differs in no way from degenerative joint disease, current practice excludes such cases and labels them simply* "degenerative disease secondary to trauma[4]" or "late (secondary) traumatic osteo-arthritis.[50]" Thus, in the tabulations of diagnoses from the army's two arthritis centers, no cases are listed under the general term "traumatic arthritis." In the classification approved by the American Rheumatism Association for inclusion in the "Standard Nomenclature of Disease", the only reference to joint disease due to trauma is listed as "Arthritis due to direct trauma."[50] (See Chapter 1, p 16)

By common usage, joint lesions resulting from acute trauma have been labeled "traumatic arthritis." It is more correct, however, to call these specific joint lesions secondary to acute trauma, inasmuch as this term includes sprains, strains, internal derangements, fractures into joints, bursitis, traumatic synovitis, hemarthrosis, lipohemarthrosis, etc At times, however, cartilage may be damaged sufficiently to cause a scar. Sufficient reaction may form about this site from repeated motion and use, so that enough excess osteoid and scar tissue results to make the joint function abnormally, eventually late secondary traumatic osteo-arthritis occurs. Such injuries may be severe (a blow or fall) or may seem so trivial that they are forgotten. Degenerative joint disease may develop following the introduction of a foreign body (such as a needle, thorn or bullet) into a joint [36] Often repeated roentgengraphic determinations may help to determine whether a progressive articular lesion has resulted from the initial trauma [10]

It must be kept in mind that *trauma may precipitate other forms of arthritis* (rheumatoid, tuberculous, syphilitic, pyogenic, gouty, etc),[49] the traumatized joint often being only the first joint involved. Also, any form

7. As pain lessens, resisted exercises are begun and deeper massage is used. Resisted exercise may be performed by the patient sitting on the edge of the table and resisting extension of the affected leg by crossing the other foot in front of it.
8. The physician must be sure that the patient is wearing shoes which fit his feet and will not throw the leg out of balance when unrestricted walking is permitted.

Moschowitz[77] has described an *inflammation of the sartorius bursa* which has not been noted previously. In his 25 cases, the disease occurred almost exclusively in women. The characteristic history was as follows: there was pain in the knees only on ascending or descending stairs, walking on the level caused no pain. Flexion or extension of the knee joint by the examiner was not painful and the joint was not tender. An area of tenderness was found at the insertion of the conjoined tendon of the sartorius, semitendino-

identical with "chronic strain of the medial ligament." No specific treatment was recommended; best results were obtained by a reduction in weight. Procaine injections into the tender area are worthy of trial, at least for symptomatic relief

Strapping for Sacro-iliac and Lumbosacral Sprains.[81]—Strips of adhesive 3 inches wide are placed transversely around the body extending for at least 2 inches beyond the crests of the ilia, the lower strip is applied first across the lower portion of the sacrum. These strips overlap each other by about one-half of their width; the highest strip comes just below the level of the lower rib margins.

A second layer of strips is applied obliquely, posteriorly overlapping each

adhesive anteriorly and to reinforce the strapping every 6 or 8 inches posteriorly.

Industrial Backache.—Backaches in industrial workers must be handled cautiously. There are few conditions which harass the industrial physician more than this sym

fuses the picture.

of coincidental anc

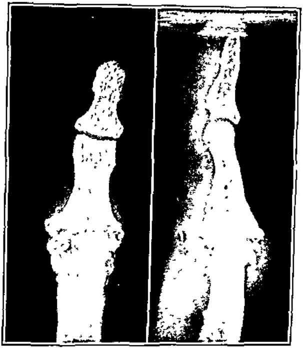
should be emphasized that in individuals over the age of fifty, changes of degenerative joint disease are present almost invariably and they may have little or nothing to do with the acute symptoms

Kidner lists the following common types of backache:[87]

(a) injury to the erector spinæ muscle, as may occur following the carrying of a heavy object,
(b) lumbosacral or sacro-iliac strain,
(c) crushed or impacted fractures of the vertebral bodies (most frequently noted at the junction of the thoracic and lumbar spine);
(d) injury of the intervertebral disks;
(e) spondylolisthesis.

of joint disease (but especially degenerative joint disease) may be aggra-

to produce acute inflammation (synovitis) of the affected joint, namely, pain, swelling, effusion and dysfunction, (2) the traumatized joint must be the only one showing such inflammation, (3) it should be established



that prior to the alleged injury articular function was normal (but the patients statement does not necessarily constitute proof); and (4) progressive articular changes may occur and in time be demonstrable radiographically (usually three to six months)

... may result in specific types of joint ... de weaver's bottom, ... layers), housemaid's ... elbow, golfer's arm, ... following: weaver's ... housemaid's knee—prepatellar bursitis; glass arm—subdeltoid bursitis or osseous deposit (see below).

He states that undoubted evidence of organic changes include: muscular spasm, sensitivity to pressure at the site of the lesion, constancy of symptoms and signs, limitation of motion appropriate for the lesion, and roentgen and neurologic findings.

In recent years many back injuries have been proven to be protrusions of intervertebral disks. Some physicians attribute most, if not all back symptoms in patients with spondylolisthesis to such protrusions. Many cases previously regarded as sacro-iliac or lumbosacral strains also have been found to have this condition. Experiences in military hospitals in World War II, however, indicate that probably too many cases of backache have been attributed to herniated disks in recent years. In many cases surgery has not relieved symptoms. This subject is covered more fully in the chapter on "Low Back Pain."

Realizing the seriousness in cost and in work lost by back injuries, attempts have been made recently to prevent such injuries by proper instruction. In one steel plant, there were 190 claims among 3700 workers in a single year.[72] To reduce such claims other workers were instructed how to lift heavy objects; during the next ten years an average of only five weeks yearly was lost by a group of 2200 workers.[73]

Gradual physical rehabilitation also has reduced time lost from work because of such injuries.[33, 53] Patients were given light work first, then gradually *returned to former occupations. By these methods disability was* shortened greatly.[35]

TEMPOROMANDIBULAR JOINT SYNDROME

In 1935 and 1936 Costen described a group of symptoms associated with disturbed function of the temporo-mandibular joint.[26,27] This syndrome (sometimes referred to as "Costen's Syndrome") has received wide attention since, particularly cases resulting from degenerative joint disease.[9, 66, 91, 93, 105] The symptoms listed by Costen include: headache, impaired hearing; stuffy sensation in the ears, tinnitus, earache; dizziness; nystagmus; burning or dryness of the throat, mouth or tongue; and herpes of the external auditory canal. These complaints were attributed to overaction of the joint at firs [illegible] ed
by absorpti [illegible] ne
case about [illegible]

[illegible]

pharyngeal neuralgia.

The pain may be relieved partially or completely by moving the condyle [illegible]

[illegible]

perfect repositioning of the jaw by dentures to [illegible]

Other forms of traumatic joint disease include tennis elbow—(involvement of the conjoined extensor tendon at the lateral epicondyle of the humerus or the radio-humeral bursa,[10] or periostitis of the epicondyle; possibly nipping of the synovia of the radio-humeral joint[50]); chauffeur's shoulder (degenerative joint disease of the shoulder joint from trauma due [illegible]

tilage).[57]

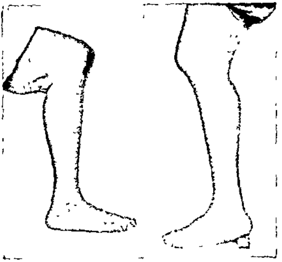


FIG 287—Bilateral prepatellar bursitis (housemaid's knee) in a scrub woman (Lewin, Orthopedic Surgery for Nurses, courtesy of W B Saunders Company)

The effect of trauma upon articular structures depends upon the type, severity and duration of the injury, as well as the reaction of the individual **The period between the injury and the production of traumatic joint disease may vary from days to several months** Pain and limitation of motion may persist for years following a single strain or contusion, even without obvious anatomic change[39]

Pathological Appearance—The pathological reaction resulting from trauma varies with the articular part affected Injury produces a two-fold effect on joints: [illegible]
or laceration of
detachment of
trauma

The **primary pathologic reaction** is a synovitis. The synovia, however, rarely is affected alone Occasionally a direct blow or contusion produces "simple traumatic synovitis," often with hemorrhagic effusion at first[65] The hemorrhagic effusion changes to serous, amber-colored, limpid fluid, usually within two to three weeks Cytologic examination of the effusion reveals a variable number of red blood cells and from 50 to 1000 white blood

WOUNDS OF JOINTS

Cope has discussed wounds of joints from a civilian and military viewpoint[25] It is usually easy to ascertain the type of wound. The **high velocity sharp-pointed bullet** may leave only one small puncture where it has entered, in other cases, one may see the wounds of entry and exit The **exit wound** may be far from the joint in another part of the limb in the case of a bullet with an oblique line of trajectory. An antero-posterior and lateral x-ray should be taken before operative treatment is used in order to tell what damage has been done to the bones

First aid treatment in any joint wound includes protection from further external infection and immobilization of the joint. Antitetanus serum

bandaged to the body or the lower limb splinted by the good limb

In the civilian type of penetrating wound (as by a blunt or pointed instrument piercing a joint), some infection practically always is carried in. If the tissues are not disturbed, they usually can deal with this. Unless one is sure that the instrument was perfectly clean, it is advisable to excise the edges of the wound down to the synovial sac, and place a narrow drain down to but not into the joint An adequate splint (or plaster cast) should then be applied Antibiotics and/or sulfonamides should be administered in adequate dosage If no fever ensues, the splint may be removed in several

around the knee joint before suturing them

In severe infections with organisms amenable to penicillin therapy (particularly in hemolytic streptococcal and staphylococcal infections), *penicillin* **should be given intramuscularly in a dosage of 50,000 units every four hours day and night Repeated aspirations of infected joints may be necessary,** in some cases it will be necessary to make a free incision for drainage, the drainage tube should not be placed into the joint

Modern bullets traveling at a high velocity may go through a joint causing

these cases are seen several hours after the injury, it is best not to interfere surgically if no untoward symptoms have developed One may excise the wounds of entrance and exit down to the synovial sac if these are seen very soon after their infliction

Considerable bloody effusion may occur in a joint cavity with loose bony fragments if a bullet traverses a joint and seriously injures the bony articular surfaces.

Berry[13] and Churchill,[21] in reporting on war wounds in World War II, have stressed **thorough débridement and wide exploration of joint wounds or compound fractures communicating with joints** The joint should be irrigated

cells (in the absence of frank hemorrhage), of which only few are polymorphonuclear leukocytes. In severely injured joints, hemarthrosis usually occurs It was present in 93 per cent of Mauck's 416 cases of injured knees.[65] Hemorrhage into joints may occur, however, without severe trauma.[84] In severe injuries which tear the synovial membrane, fat may be extravasated from the juxta-articular fat pads, creating **lipohemarthrosis.**[81] Such an injury should stimulate a search for obscure fractures not recognizable on routine roentgenographic projections [92]

When articular structures other than the synovial membrane are injured, pathological changes resembling those of degenerative joint disease result almost invariably. Such changes are hastened by overweight (in weight-bearing joints), overuse or the continued presence of loose bodies. Some believe that if effusions are not removed the synovial membrane becomes



Fig 288.—*Traumatic*
bodies due to a gun sh
groin one year before
and the bullet was not t

permanently thickened and irregular, with the late formation of **hypertrophic or villous synovitis.**[76]

In traumatic tears of a joint capsule, a serofibrinous exudation occurs which may lead to the formation of periarticular adhesions. The latter result also from injuries of the periarticular structures themselves. Ad-

mobilized with plaster. After forty-eight hours the joint is reaspirated and penicillin instilled; this process is repeated at forty-eight hour intervals

nd the above antibiotic

By such measures
penetrating wounc
fourteen days old
time [13]

Altemeier advises a program of *intra-articular* and *parenteral penicillin* for four to five days. If parenteral penicillin has been given for more than twelve hours before aspiration, no intra-articular penicillin is given. If seen before antibiotic administration the joint is aspirated

are instilled The amount of diluent used should be approximately one-fourth to one-half the amount of fluid removed (or 10 cc. if only a small amount is obtained). Intramuscular penicillin is given in a dosage of 50,000 units every four hours *if the contamination is minimal and no great amount of mixed infection is present.*

When contamination is heavy and mixed infections of Gram-positive and negative, aerobic and anaerobic bacteria are anticipated (such as from auto and airplane accidents, *etc*), 100,000 units of penicillin and ½ gram of streptomycin are given intramuscularly every eight hours When mass casualties occur and nursing care is a factor, 500,000 units of penicillin and ¾ gram of streptomycin are administered every twelve hours.

Ordinarily, four to five days' treatment is sufficient. If beneficial results are not obtained by this time, other antibiotics should be tried. If the patient is sensitive to penicillin, other antibiotics should be used from the outset.

In severe lacerated wounds with badly injured articular surfaces which may be broken into fragments and contaminated by various kinds of débris, amputation may be required if the main vessels of the limb are destroyed. One should always give the joint a chance to recover if the main vessel is intact. Antitetanus and anti-gas-gangrene serum as well as antibiotics should be administered and shock must be treated; when the patient is in condition, the wound is cleaned and infected tissues are removed A portion of the synovial membrane is left open for drainage and the limb is encased in plaster. Penicillin and/or sulfonamides should be administered in adequate dose.

The use of sulfonamides, penicillin, tetanus toxoid and other indicated measures have greatly decreased the necessity for amputation in these

patient's general condition (as evidenced by fever, wasting and general

hesions in these structures and particularly in plications of the joint capsule may produce stiffness of a joint with muscular spasm and eventually contractures.

Symptoms and Signs.—Traumatic joint disease usually is monarticular. Symptoms include **pain,** particularly on motion, **swelling** and **limitation of motion.** Objectively one finds localized **tenderness, swelling** (often with frank effusion), **limited motion,** frequently **ecchymosis, muscular spasm** and **crepitus** on motion When effusions are recurrent one should suspect internal derangements or other forms of arthritis

Treatment.—In acute traumatic joint disease, rest, heat, and the removal

mend aspiration as soon as possible to prevent the formation of dense

hypermobile cartilages and subsequent locking and damage [65] Some authors, however, do not favor routine aspiration unless the fluid persists.

A **sponge compression dressing** has been recommended for patients with traumatic synovitis of the knees Forrester applies a protective cotton pad

ssing of sheet wad-

to fit the contour

ize bandage The

t, the patient may continue

Joint Disease· This includes· (1) prevention of the disease by restoring normal alignment and obtaining accurate anatomic reduction in fractures, in the case of joint fractures, open operation may be necessary to obtain anatomic reduction, (2) medical treatment (by weight reduction in obese individuals with traumatic lesions of weight-bearing joints), (3) physical therapy (rest, heat, massage and exercises); and (4) surgery (including removal of foreign bodies in joints)

1. Contraction of the quadriceps for two minutes every hour while lying on a couch
2. Alternate flexion and extension of each knee (20 times twice a day) with the knees bent and the legs hanging over the edge of a couch
3. Lying flat in bed, raise the extended leg by flexing the hip Abduct and then adduct the thigh at the hip.
4. As improvement occurs, "bicycling" of the legs
5. Later, use weight-bearing exercises, such as raising the heels, bending the knees and straightening upright while holding the back of a chair

appearance) makes it obvious that the patient's general strength is being definitely but steadily undermined, amputation may be necessary. It is the consensus of present opinion that with newer methods of chemotherapy, the surgeon now must really prove that amputation is a necessity and that further sulfonamides or antibiotics will not be adequate assistive treatment

CAISSON DISEASE OF BONE

Caisson disease may cause bone and joint pains, particularly in the lower extremities (mainly knees) Usually it is monarticular. Acute symptoms of faulty decompressions are known as "the bends." While such disease usually is found in divers, identical lesions recently have been reported in persons never subjected to abnormal atmospheric pressure or trauma.[100,101] In these cases the lesions were attributed to fat emboli, infarcts, septic foci and peripheral vascular disease. Acute pain in bones and joints similar to that in Caisson disease but without infarcts or aseptic necrosis of bone have [illegible] h pain [illegible] to an accumulation of nitrogen, causing "gas embolisms" in the nutrient arteries [illegible]

cartilaginous loose bodies may be present Characteristic findings include medullary calcifications and disturbance of the lamellar structure of the ends of the large long bones.[88] Roentgen examination reveals multiple distribution of infarcts in the medullary bone with few cortical changes

ACUTE BONE ATROPHY (PAINFUL OSTEOPOROSIS; CAUSALGIA)

This condition in most cases is post-traumatic. Frequently, however, such trauma may be minimal or absent. It has been noted following contusions, sprains, fractures, and mild or severe burns It has been observed also after relatively minor surgical procedures in extremities and after various acute and chronic diseases of bones, joints, and tendons Recently, a similar clinical syndrome was reported as a complication in 2 per cent of hypertensive patients treated with potassium thiocyanate.[51]

The cause of this condition is unknown. Apparently, it is a vasomotor disturbance characterized by hyperemia in bone, increased vascularity, thinning of bone lamellæ and filling of the interlamellar spaces with fibrous tissue Hyperemia in soft tissues produces duskiness, edema, local heat, periarticular thickening and limited motion[60] Rapid muscular weakness and wasting accompany the process Eventually, the part usually shows cold, clammy skin, cyanosis, desquamation, atrophy of the skin and subcutaneous tissues and delayed wound healing. Roentgenograms reveal patchy atrophy of bone after approximately four to six weeks

Recently, De Takats offered an explanation for this condition.[31,32] He stated that Sudeck's atrophy, Leriche's post-traumatic osteoporosis, Weir Mitchell's causalgia, peripheral trophoneurosis, reflex dystrophy and chronic

Joints which are the seat of traumatic disease may be splinted by Castex or molded plaster o
the acute phase of
full painless motior

In the treatment of *chronic traumatic joint disease*, Hench advises rest and avoidance of any operative trauma; physical therapy; correction of posture; use of proper shoes, supports and exercises for the feet; weight reduction (if indicated and in some cases, the use of splints or roller bandages, and special corsets or braces.[41] He stresses prophylaxis against this disease by rest periods and insulating traumatized joints by leather pads, etc.

SPRAINS

A sprain may be defined as a stretching or tearing of the supporting ligament about a joint by forced movement beyond its normal range. Miltner and his co-workers found that after experimental sprains in rabbits, the tissues were normal grossly in four weeks, but microscopic inflammation persisted

joints Usually in a sprained ankle, rotation of the calcaneum occurs in pronation, causing the thrust of the body weight to fall upon the lateral

bility of a fracture. If a bone breaks, the ligaments as a rule are not torn.

Clinical Manifestation.—The initial pain of a sprain often is excruciating, even nauseating, and the patient may turn white, develop cold, clammy perspiration and faint The soft parts become swollen within a very short time. Some ecchymosis may develop later but as a rule is less extensive than with a fracture. Tenderness usually is diffuse in contrast to the more localized tenderness over fractures. Abnormal mobility and crepitus are absent. However, it is wise to X-ray the part if the injury is severe for it is almost impossible to deny the possibility of a fracture in these sprains without roentgenogram

In addition to torn ligaments, frequently small blood vessels also are
form a hematoma, this is
st of the ensuing pain
which develops from
the confining of this fluid to a small space accumulation and the disuse resulting from pain on motion lead to stiffness of the part.

Treatment.—For simple sprains *procaine* injection followed by *adhesive strapping* probably is the best treatment. Ochsner has diagrammed the technic of strapping various areas [81] For easy removal from the skin he advises rubbing about a teaspoonful of cold cream into the skin areas to

esthetics injected into
sprains.[56] This treat-
The effect from such
injections persists long enough to permit some removal of fluid, sufficient in amount to relieve the tension in the injured part. The relief of pain per-

traumatic edema all ... [illegible]

due to chronic irritation of sensory pathways producing cutaneous hyperalgesia, burning pain and a local increase in blood flow. Three sensory levels are recognized—a peripheral, spinal and cortical The lesion becomes worse and more intractable the higher it ascends Peripheral trauma activates the lowest neuron, producing vasomotor reflexes which under certain conditions are predominately vasodilator This produces increased capillary pressure and *stimulation of sensory receptors with the resultant* throbbing, burning pain of causalgia

Lewis has postulated the secretion of a pain substance at the termination of nerve fibers belonging to the posterior root system [86] This substance may be acetylcholine or histamine though it is not clear which it may be since both have been found. In irritative lesions in any of the three sensory neurons a group of fibers is activated which secretes pain-producing vasodilator substances at nerve endings In the causalgic state there is an [illegible]

mistreated so that the first neuron lesion may ascend to the second level [illegible]

pain may develop. Contractures and ankylosis occur.

It is obvious from this description that **the feature of osteoporosis has been overstressed.** It does not make its appearance for some four to six weeks When the osteoporosis is at its height the syndrome may be subsiding

Treatment.—Early treatment is most important An understanding of which sensory neuron is affected is essential.[31,32] In the early stages, the site of trauma should be **immobilized** and **thoroughly infiltrated daily with 1 per cent procaine solution.** This blocks sensory stimuli and inhibits secretion of the vasodilator substances. Physical therapy also should be given

If pain is not relieved and particularly when osteoporosis has appeared, [illegible] of the paravertebral sympathetics is recommended In a series [illegible]

days, **sympathectomy or sympathetic ganglionectomy** is indicated Both Evans and De Takats warn that **no relief may be expected of sympathectomy unless temporary relief has been attained by block.** Recently, following the experimental work of Acheson and Moe,[1,2] the parenteral use of **tetraethylammonium bromide and chloride** has been *advised in causalgic states* [illegible] Berry

The drug may be given either intramuscularly or intravenously Following injection by either route, the blood pressure drops abruptly, often as much

trauma was present in 25. **Pathological findings** were those of an aseptic necrosis of bone. At times there was a central area of necrotic bone, at other times an irregular mass of ossifying cartilage with foci of necrosis at the periphery. A *thick layer of hyaline cartilage* was present on the articular surface, a thin layer of fibrocartilage on the opposite surface.

Roentgenograms may be negative for as long as six months after a causal injury; when positive, a sharply defined cavity is found from which a sequestrum of bone arises This sequestrum may be in the cavity, may be covered by unbroken cartilage or may have been extruded into the joint space.



FIG. 290 —Osteochondritis dissecans (Holmes and Robbins.)

[illegible] than 85 per cent of cases; the elbow

[illegible]

in a terminal phalangeal epiphysis of [illegible] involvement of the tarsal navicular [illegible] reported recently[83] Almost every growth center may be involved in **"osteochondritis of growth centers."** Varieties of this condition often are regarded as separate diseases (Osgood-Schlatter disease; Legg-Calve-Perthes' disease, etc.).[85] It is believed that insufficiency of the blood supply to the involved epiphysis may play a more important causal rôle than trauma

The possibility of this disease in a joint should be considered when [illegible] the usual conservative therapy in [illegible] t may be suggested by recurrent [illegible]rness, crepitus and wasted quadriceps *following an injury (to the knee), etc.*

Conservative treatment (rest and immobilization) is recommended for mild cases.[89] In most cases, however, arthrotomy with removal of the

loose fragments is advisable,[43] it should be followed by physical therapy (heat, massage and graded exercises).

Epiphysitis of the femoral head was first described by Legg in 1909. Levy and Girard studied 102 cases, of which 91 per cent were in males. In 10 per cent the disease was bilateral. The age of the patients when first seen (usually soon after the onset) varied from three to thirteen years (averaging 8.6 years). The average case ran a course of about two-and-a-half years with proper treatment, as long as four years with improper treatment.

SYNOVIAL CYSTS OF THE POPLITEAL SPACE; "BAKER'S CYSTS"

In 1877 and 1885 Baker described synovial cysts in the leg with disease of the knee.[7] He stated the cysts resulted from osteoarthritis. Recently,

The cause of these cysts is unknown. Trauma is blamed by some. Meyerding and Van Demark noted rheumatoid or osteoarthritis in 7 of their 15 cases.[74]

The lesion consists of a swelling located in the popliteal space. It may fluctuate in size due to passage of fluid into the joint (and vice versa) or possibly from variations in the secretory or absorptive powers of its walls. They may be from 8 to 10 cm. long and 5 to 7 cm. wide.[74] With enlargement they may extend down the back of the leg as far as the Achilles tendon. The condition may occur at any age, Haggart, however, states that those in children usually consist of semimembranosus bursae, those of adults usually posterior herniations.[44]

Few symptoms are produced by this condition. Mild aching and stiffness may occur. Often the presence of the swelling is noted without other symptoms. The cyst may appear soft on flexion (or may disappear) and be firm on extension of the knee. Flexion may be somewhat limited. While in most cases the cysts are unilateral, occasionally they are bilateral.[84]

Transillumination of the area reveals the cystic character of the lesion.

swellings and Charcot's joints.

Opinions vary as to the exact nature and origin of these swellings but there appear to be several types: (1) a posterior herniation of the capsule of the knee,[60] (2) hernial protrusion of synovial membrane from the superior tibiofibular joint, (Whalley)[107] (3) semimembranosus bursitis,[18] (4) enlarged semimembranosus bursa,[18] (5) either posterior herniation of articular capsule (63 per cent of cases) or semimembranosus bursa enlarged by "hyperplasia-fluid distention (37 per cent),[44] (6) generally "herniation of synovial membrane through the posterior part of the capsule, or, less commonly, enlargement of either the semimembranosus or the medial gastrocnemius bursa by an escape of synovial fluid from the knee into either

of these bursae via the normal anatomic connections between knee and bursae."[74]

There is no general agreement of the **pathological appearance** of these cysts Meyerding and Van Demark believed them lined by endothelium, Haggart, by mesothelium similar to synovial membrane, Whalley described a hyaline fibrous wall with no endothelial or epithelial lining Most authors believe that some element of inflammation often is present

Treatment is essentially surgical. While possibly a few cases improve by rest and bandaging, this improvement rarely is permanent Careful dissection and ligation of the pedicle is the treatment of choice. The cyst may recur however if the dissection is not thorough. The use of sclerosing solutions is not recommended [60]

PELLEGRINI—STIEDA SYNDROME

Pellegrini—Stieda disease may be defined as para-articular calcification at the medial aspect of the knee joint. The exact nature of this syndrome

epiperiosteal hematoma,[64] calcification of the bursa between the tibial collateral ligament and the capsule superior to the medial meiscus;[17] calcified tendon of the vastus medialis,[16] "peritendinitis calcarea;" calcific ligamentitis of the tibial collateral ligament;[40] and calcified hematoma in soft tissue close to the medial condyle of the femur.[4]

Symptoms of this syndrome include stiffness and pain above the knee on the medial aspect Swelling may be present Diagnosis is made by roentgenogram (See Figure 33 p 97.)

to trauma. Russell and s.[89] Hench mentions the Clinic) have studied 52 *post-traumatic calcifica-*

Treatment.—Some cases heal spontaneously. (Russell and Smith[89]). Physical therapy generally controls symptoms and is followed at times by *disappearance of the calcific deposits* [79] Roentgen therapy has been advised by some,[40] surgical excision if conservative measures do not help [64]

GANGLION

A ganglion is a cystic swelling cntaining thick mucoid material, occurring usually along tendon sheaths or joint capsules It is found most commonly in the wrist. It occurs anists, typists and the swelling and occasionally slight ... of ganglia is quite thin and closely resembles that of joints or bursae

The origin and nature of ganglia are in dispute They have been considered as herniations of the synovial membrane *through tears in the*

but not all cases.

Numerous treatments have been proposed. Cherry and Ghormley state that 58 per cent of ganglia disappear spontaneously.[90] Treatment includes pressure bandages; rupturing the ganglia with a blow (as from a heavy

recurs. Cherry and Ghormley obtained good results from complete surgical excision. Hydrocortisone has been injected into the ganglion in three cases[32, 90] recently, with complete disappearance of the lesion resulting in all

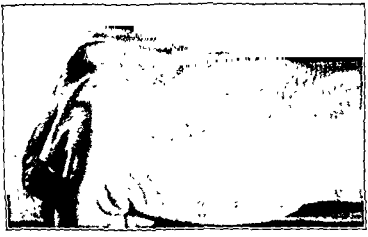


FIG 291.—Ganglion arising from extensor tendon sheaths—oval and flattened cystic mass on dorsum of hand (Brunschwig's Surgery, courtesy of C V Mosby Company.)

PERITENDINITIS CREPITANS

There is a palpable and often an audible crackling crepitus in some cases

or hydrocortisone injection of the tendon sheath is sometimes worthy of trial.

INJURIES TO MUSCLES AND TENDON

Haldeman and Soto-Hall discussed certain features of injuries to muscles and tendons.[45] Features which they found very suggestive of rupture of muscles included.

1. A history of a sudden sharp pain or snapping sensation occurring during violent muscular effort.
2. Inability to perform certain definite movements following this

3. The diagnosis is more certain if one sees the appearance of a defect in the belly of the muscle or in the tendon with subsequent ecchymosis.

4. Roentgenogram of soft tissues often reveals a defect in the shadow cast by the muscle; in some cases, a small chip of bone is seen attached to a tendon which has been torn from its insertion.

5. Electrical stimulation with the faradic current if applied to the muscle will cause it to contract and produce pain at the site of a tear in the muscle or tendon.

6. Local injection of procaine will eliminate pain and will permit one to test muscular function in those cases in which the diagnosis is difficult.

The supraspinatus muscle or tendon is the most likely to be torn in the upper extremity and the quadriceps is the most vulnerable in the lower extremity.[45] The average age of patients with tears of muscles or tendons was forty-two years, corresponding to the period in which the greatest activity is associated with degenerative changes.

The most common etiologic factor is acute trauma. Sudden application of a stretching force to a muscle that is contracting strongly is the most common mechanism producing injury to a muscle or tendon. Spontaneous rupture is relatively rare.

In 1922, Grassheim reported that in 500 cases of injury to muscles and tendons, the rupture occurred most frequently in the muscles of the calf, extensors of the leg, biceps, Achilles tendon and extensor of the thumb.[45] In recent years, other frequent sites of rupture of muscles have been the supraspinatus, triceps, rectus abdominis, adductors of the thigh and extensors of the fingers.[45]

McMaster's experiments on the breaking weight and site of rupture of a preparation of muscle and tendon showed that normal tendon does not rupture but that the rupture occurs at the attachment of tendon to bone, the musculotendinous junction or through the muscle substance.[69]

Codman has emphasized the importance and frequency of partial or complete *tears of the supraspinatus tendon*.[22] Some individuals believe that Codman confused certain anatomic changes in the shoulder that are the result of senile wear and tear with purely traumatic lesions. Meyer described attritional changes in the shoulder with longitudinal fibrillations or fraying of the supraspinatus tendon.[73] Supraspinatus tears are rather infrequent clinically.[95] (See also under Painful Shoulder p. 944.)

Rupture of the long head of the biceps has been discussed by numerous authors.[45] The biceps tendon becomes frayed out (with advancing years) in the bicipital groove and over the head of the humerus where it is subjected to constant friction

Rupture of the long head of the biceps is followed by marked weakness of the flexor muscles of the elbow and is often accompanied by pain over the anterior aspect of the shoulder joint or over the bicipital groove The belly [illegible]

downward through the sheath and present in the belly of the muscle. The [illegible]

snap which can be felt and heard (over the occipital groove). Weakness of the arm and localized tenderness result from permanent dislocation of

[illegible]

The **rhomboideus major or minor muscles** may be strained by a sudden incoordinated movement of the shoulder; this occurs frequently in industrial practice. These muscles arise from the ligamentum nuchæ and the spinous process of the seventh cervical to the fifth dorsal vertebrae and insert into the vertebral border of the scapula. Contraction of these muscles draws this border of the scapula upward

Following injury to the rhomboid muscles there is a localized tenderness between the mid-dorsal spine and the scapula; pain occurs at this point if the shoulder is passively flexed forward markedly or if it is extended against resistance. Treatment consists of partial immobilization of the scapula by drawing it backward and upward with adhesive Physical [illegible] ld be begun when the signs of

[illegible] processes and inserting on the scapula or humerus include the **latissimus dorsi and trapezius muscles**, strains of these muscles give a clinical picture which is similar to injury to the rhomboids. Therapy is the same

Rupture of the rectus abdominis muscle may at times be mistaken for [illegible]

the epigastric vessels The hematoma may require evacuation surgically.

Rupture of the **quadriceps muscle** or tendon is very common This occurs at the point of attachment of the tendon to the patella or at the musculotendinous junction; occasionally, a tear may occur through the purely tendinous or muscular portions

The usual cause of this condition is a direct blow, such as falling on a flexed knee. The injury occurs in both young and aged It is diagnosed by the history and by the inability of the individual to actively extend the knee, in addition to the hiatus which is seen in the tendon or muscle. The gap in the muscle may be seen by a lateral soft-tissue roentgenogram, the superior border of the patella usually tilts anteriorly Partial tears of the muscle may leave only a depression in the muscle substance without permanent disability These usually heal readily following splinting of the leg in full extension for three or four weeks, with gradual mobilization following this Complete tears of the quadriceps muscle or tendon should be repaired surgically.

Strong contraction of the quadriceps muscle may pull the tubercle from the front of the tibia (**Osgood-Schlatter's disease** or **Schlatter's sprain**). [illegible]

patella sags downward following rupture of the quadriceps tendon above the patella; there is loss of the power of leg extension and a dimple may be seen above or below the edge of the patella.

Partial rupture of one of the calf muscles has been termed "tennis leg;" this usually involves the plantaris muscle, but may affect a belly of the gastrocnemius. Symptoms include a snap (audible or palpable) with sudden burning pain in the calf during a strong muscular contraction. The pain may extend to the popliteal space; it is aggravated by passive dorsiflexion of the ankle. Treatment includes adhesive strapping to immobilize the ankle in plantar flexion for several weeks, following which baking, massage and exercises are used.

Violent exercise (as in boxers, sprinters, etc.) may result in injury to the Achilles tendon which is usually ruptured at the musculotendinous junction. In complete tears, there is a marked depression (from separation of the fibers of the tendon); the foot cannot be flexed in the plantar direction actively against resistance. Partial rupture may produce tenderness and ecchymosis over a depression in the tendon. In the latter, treatment consists of adhesive strapping or a cast to immobilize the foot in plantar flexion. Complete tears require surgical intervention.

McMaster reviewed the literature and found 27 instances of late rupture of the extensor pollicis longus following Colles' fracture.[63] From one to three months may elapse before its appearance. The lesion is caused by chronic trauma from the irregularity of the bone over which the tendon moves.

The so-called *"baseball finger"*—an extremely common and troublesome injury—is caused by rupture or detachment of the extensor tendon at its insertion Immediate hyperextension of the terminal joint for at least six weeks will often avoid deformity; later operative repair is frequently unsuccessful.

Herniation of a muscle occurs occasionally through its sheath. This results most often from a direct blow. It may be repaired with fascial strips.

Injections of procaine may relieve the pain of muscular injury, fibrositis or acute tendinitis [36,61]

Bennett discussed the *"sore arm" of the professional baseball pitcher,* dividing the shoulder lesions into an anterior and a posterior group [11] The anterior group included traumatic irritation, fraying of the supraspinatus and biceps tendons associated with constant rotation of the humerus, similar traumatic irritation of the subdeltoid and subacromial bursae, irritation of the coracoid [illegible] the head of the hum[illegible] "follow-through" and inflamma[illegible]s in the various [illegible] is region. Conservative [illegible] effective in [illegible] rest, local heat, and remo[illegible] [illegible]dent focal inf[illegible] of the general health.

Th[illegible] characteristic [illegible] an exostosis [illegible]lop at [illegible] inferior [illegible] fossa, [illegible] of the tricep[illegible] capsule. [illegible] [illegible]er of the [illegible]osit in[illegible] and pro[illegible] ter[illegible] [illegible]e, with [illegible] in th[illegible] [illegible] under [illegible]

these individuals of rehabilitation of the shoulder so that it can withstand this strain

Elbow lesions which may occur in the pitcher include loose bodies in the olecranon and coronoid fossæ and other osteochondritic changes These produce few symptoms other than interference with complete extension of the elbow. Surgery usually is successful so that the pitcher's career is not affected

BIBLIOGRAPHY

1 ACHESON, G H, and MOE, G. K.: Jour Pharmacol and Exper Therap, 84, 89, 1945
2 ——— *Ibid*, 87, 220, 1946
3 AITKEN, A P.: New England Jour Med, 232, 105, 1945
4 ALLEN, W E, JR: Jour Nat Med Assn, 37, 164, 1945
5 ALTEMEIER, W. A: Personal Communication

10 BENNETT, G A Clinics, 1, 1448, 1943
11 *BENNETT, G E: Jour Am Med Assn, 117, 510, 1941*
12 BERNSTEIN, M A, and FISHER, H E Surg Clin North America, 17, 363, 1937
13 BERRY, F B Quoted by Cole and Elman [24]
14 BERRY, R L, CAMPBELL, K M, LYONS, R. H, MOE, G K, and SUTLER, M R Surgery, 20, 525, 1946
15 BIERMAN, H. R Washington Univ Med Alumni Quart, 6, 160, 1943
16 BORAK, J: New York State Jour Med, 45, 725, 1945

int Surg, 25, 121, 1943

2, 319, 1941

npany, 1934
23 COHEN, H H Bull Hosp Joint Dis, 4, 86, 1943
24 COLE, W H, and ELMAN, R Textbook of General Surgery, New York, D Appleton-Century Company, p 1097, 1948
25 COPE, V. Z: Brit Med Jour, 1, 648, 1942
26 COSTEN, J B Arch Otolaryng, 22, 554, 1935
27 ——— Jour Am Med Assn, 107, 252, 1936
28 COTTRELL, J C. Jour Am Med Assn, 124, 81, 1944.
29 COZEN, L Physiotherap Rev, 21, 151, 1941
30 COZEN, L, and HOLLOMBE, B S Surgery, 8, 468, 1940
31 DE TAKATS, G: Arch Neurol and Psychiat, 1, 318, 1943
32 ——— Jour Am Med Assn, 128, 699, 1945
33. DOUGHERTY, W: Indust Med, 11, 473, 1942
34. DURCHAK, E T Arch Phys Therap, 23, 95, 1942
35 ECKER, A: Jour Am Med Assn, 119, 128, 1942
36 Editorial Rheumatism, vol 2, No 6, 1942
37 EVANS, J A: Jour Am Med Assn, 132, 620, 1946
38 FORRESTER, C R G Am Jour. Surg, 28, 145, 1935
39 FOX, R F. Jour State Med, 41, 263, 1933
40 FRANK, C H. Tri-State Med Jour, 15, 2925, 1943
41. FRIEDLICH, A L, JR, CHAPMAN, W. P, and STANBURY, J B New England Jour Med, 238, 629, 1948
42. GHORMLEY, R K Surg Clin North America, 23, 946, 1943
43 [illegible]

Med Assn, 104, 2319, 1935
, 78, 556, 1949

Partial **rupture of one of the calf muscles** has been termed "**tennis leg**;" this usually involves the **plantaris muscle**, but may affect a belly of the gastrocnemius. Symptoms include a **snap** (audible or palpable) with sudden burning pain in the calf during a strong muscular contraction. The pain may extend to the popliteal space; it is aggravated by passive dorsiflexion of the ankle Treatment includes adhesive strapping to immobilize the ankle in plantar flexion for several weeks, following which baking, massage and exercises are used.

Violent exercise (as in boxers, sprinters, etc.) may result in **injury to the Achilles tendon** which is usually ruptured at the musculotendinous junction In complete tears, there is a marked depression (from separation of the *fibers of the tendon), the foot cannot be flexed in the plantar direction* actively against resistance. Partial rupture may produce tenderness and ecchymosis over a depression in the tendon. In the latter, treatment co

fle

of the **extensor pollicis longus** following Colles' fracture [63] From one to three months may elapse before its appearance The lesion is caused by chronic trauma from the irregularity of the bone over which the tendon moves.

The so-called *"baseball finger"*—an extremely common and troublesome injury—is caused by rupture or detachment of the extensor tendon at its insertion. Immediate hyperextension of the terminal joint for at least six weeks will often avoid deformity, later operative repair is frequently unsuccessful.

Herniation of a muscle occurs occasionally through its sheath. This results most often from a direct blow It may be repaired with fascial strips.

Injections of procaine may relieve the pain of muscular injury, fibrositis or acute tendinitis [36,61]

Bennett discussed the *"sore arm" of the professional baseball pitcher,* dividing the shoulder lesions into an anterior and a posterior group [11] The **anterior** group included traumatic irritation, fraying of the supraspinatus and biceps tendons associated with constant rotation of the humerus, similar traumatic irritation of the subdeltoid and subacromial bursae, *irritation of the coracoid bursa* by the head of the humerus in the "follow-through" and inflammatory processes in the various bursae in this region Conservative treatment usually is effective in these; this includes rest, local heat, and removal of any evident focal infection and improvement of the general health.

The characteristic **posterior** lesion is an exostosis which may develop at the postero-inferior border of the glenoid fossa, probably due to repeated ... and posterior capsule This lesion may seriously

...sule and its
...tation of the
...ion. Symp-
...nnot assure

these individuals of rehabilitation of the shoulder so that it can withstand this strain.

Elbow lesions which may occur in the pitcher include loose bodies in the olecranon and coronoid fossæ and other osteochondritic changes These produce few symptoms other than interference with complete extension of the elbow. Surgery usually is successful so that the pitcher's career is not affected.

BIBLIOGRAPHY

1 Acheson, G H , and Moe, G. K · Jour Pharmacol. and Exper Therap , **84**, 89, 1945.
2 ——— · *Ibid* , **87**, 220, 1946.
3. Aitken, A P.: New England Jour Med , **232**, 105, 1945
4 Allen, W E , Jr · Jour Nat Med Assn , **37**, 164, 1945
5 Altemeier, W A : Personal Communication
6 Arnulf, G , and Frieh, P · Quoted by Leriche and Arnulf [46]
7 Baker, W M Med Classics, **5**, 805, 1941
8 Balensweig, I : Surg Clin of North America, **23**, 497, 1943
9 Bauer, W H.. Amer Jour Path , **17**, 129, 1940
10 Bennett, G A Clinics, **1**, 1448, 1943
11 Bennett, G. E. Jour Am Med Assn , **117**, 510, 1941
12 Bernstein, M A , and Fisher, H E Surg Clin North America, **17**, 363, 1937
13 Berry, F. B : Quoted by Cole and Elman [24]
14 Berry, R L , Campbell, K M , Lyons, R H , Moe, G K , and Sutler, M R Surgery, **20**, 525, 1946
15 Bierman, H R · Washington Univ Med Alumni Quart , **6**, 169, 1943
16 Borak, J. New York State Jour Med , **45**, 725, 1945
17 Brantigan, O C , and Voshell, A F Jour Bone and Joint Surg , **25**, 121, 1943
18 Burman, M Jour Am Med Assn , **124**, 29, 1944
19 Carp, L Arch Surg , **24**, 95, 1932
20 Cherry, J. H , and Ghormley, R K Am Jour Surg , **52**, 319, 1941
21 Churchill, E D · Ann Surg , **120**, 268, 1944
22 Codman, E A The Shoulder, Boston, Thomas Todd Company, 1934
23 Cohen, H H Bull Hosp Joint Dis , **4**, 86, 1943
24 Cole, W H , and Elman, R Textbook of General Surgery, New York, D Appleton-Century Company, p 1097, 1948
25 Cope, V Z Brit Med Jour , **1**, 648, 1942
26 Costen, J B Arch Otolaryng , **22**, 554, 1935
27 ——— Jour Am Med Assn , **107**, 252, 1936.
28 Cottrell, J C Jour Am Med Assn , **124**, 81, 1944
29 Cozen, L . Physiotherap Rev , **21**, 151, 1941
30 Cozen, L , and Hollombe, B S Surgery, **8**, 468, 1940
31 De Takats, G . Arch Neurol and Psychiat , **1**, 318, 1943
32 ——— · Jour. Am Med Assn , **128**, 699, 1945
33 Dougherty, W Indust Med , **11**, 473, 1942
34 Duschak, E T Arch Phys Therap , **23**, 95, 1942
35 Ecker, A · Jour Am Med Assn , **119**, 128, 1942
36 Editorial Rheumatism, vol **2**, No 6, 1942
37 Evans, J. A · Jour Am Med Assn , **132**, 620, 1946
38 Forrester, C R G Am Jour Surg, **28**, 145, 1935
39 Fox, R F Jour State Med , **41**, 263, 1933
40 Frank, C H Tri-State Med Jour , **15**, 2925, 1943.
41 Friedlich, A L , Jr , Chapman, W P , and Stanbury, J B New England Jour Med , **238**, 629, 1948
42 Ghormley, R K Surg Clin North America, **23**, 946, 1943
43 Grassheim: Monatschr F Unfallh , **29**, 313, 1922
44 Haggart, G E Lahey Clin Bull , **3**, 243, 1944
45 Haldeman, K O, and Soto-Hall, R Jour Am Med Assn , **104**, 2319, 1935
46 Ha [illegible] , **78**, 556, 1949
47 He [illegible]
48 — [illegible]
49 — [illegible]

50 Hench, P S : *Ibid*, **28**, 66, 1948
51 Hinchey, J J, Hines, E A, Jr, and Ghormley, R K.. Proc Staff Meet, Mayo Clin, **22**, 275, 1947.
52 Hollander, J L, Brown, E. M, Jessar, R A, and Brown, C. Y · Jour Am Med Assn, **147**, 1629, 1951
53 Howard, N. J.. Jour Bone and Joint Surg, **19**, 447, 1937
54 Jefferson, G . Proc Roy Soc-Med (Sect for Study Dis Child), 13, 162, 1920
55. Jones, M L Wisconsin Med Jour, **44**, 1069, 1945
56 Key, J A
57 Kidner, F C
58 Kilfoy, E. J
59 Kraus, H
60. Kuhn, H H, and Hemphill, J E Radiol, **42**, 237, 1944
61 Lapidus, P. W
62 Lavner, G Am
63 Leriche, R, and
64 Levinthal, D H
65 Mauck, H P Am Jour Surg, **56**, 54, 1942
66 McLaren, J W Brit Jour Radiol, **17**, 94, 1944.
67 McLaughlin, C W, Jr Military Surgeon, **97**, 457, 1945
68 McMaster, P E Jour Bone and Joint Surg, **14**, 93, 1932
69 ——— *Ibid*, **15**, 705, 1933
70 ——— Jour. Am. Med. Assn, **122**, 659, 1943
71 Meadoff, N, and Gray, A B Med. Bull, **2**, 106, 1944
72 Means, W H · Pennsylvania Med Jour, **46**, 103, 1942
73. Meyer, A W Arch Surg, **17**, 493, 1928
74 Meyerding, H W, and Van Demark, R E : Jour. Am Med Assn, **122**, 858, 1943
75 Miltner, L J, Hu, C H, and Fang, H. C Arch. Surg, **35**, 234, 1937.
76 Moorhead, J J, and Lyall, D. Ann Surg, **117**, 140, 1943
77 Moschcowitz, E Jour Am Med. Assn, **109**, 1362, 1937.
78 Murray, H G Indust Med, **12**, 730, 1943
79 *Nachlas, O W, and Olpp, J L Surg, Gynec, and Obst, **81**, 206, 1945.*
80 Nairn, S J G Brit Jour Phys Med, **6**, 214, 1932
81 Ochsner, E H Med Rec, **145**, 294, 1937
82 O'Donnell, E D Physiother Rev, **20**, 218, 1940
83 O'Donaghue, A F, Donohue, E J, and Zimmerman, W. W · Jour Bone and Joint Surg, **30-A**, 780, 1948
84 Peirce, C B, and Eaglesham, D C Radiology, **29**, 655, 1942
85 Potter, P C Ann. Surg, **117**, 290, 1943
86. Quoted by De Takats [29]
87 Ray, M B Jour. State Med, **40**, 255, 1932

30, 189, 1943
1947
, **145**, 1941
Med, **41**, 318, 1934
935
g, **64**, 328, 1944

102 ——— Radiology, **42**, 550, 1944
103 Titus, N E . Discussion of Key, No 52
104 ——— Arch Phys Ther, **14**, 285, 1932
105. Tucker, W E : Brit Med Jour, **2**, 525, 1937.
106 Vaughn, H C. Jour Am Dent Assn, **30**, 1501, 1943
107. Whalley, N.· Brit Jour Surg, **31**, 306, 1944

CHAPTER 53

INTERNAL DERANGEMENT OF THE KNEE JOINT

By Nathan R. Abrams, M.D.

Internal derangement of the knee joint may be defined as interference with the normal mechanical function of the joint due to displacement of some constituent, or to the presence of an abnormal or foreign product within the joint. Such derangements are accompanied by a varying degree of synovial hyperemia and effusion. If the irritating body is not removed this membrane becomes permanently thickened and irregular with the late formation

[illegible]

shape gives no strength to the joint In spite of this, dislocations of the knee are uncommon. However, mechanical derangements are encountered commonly in the knee joint

Symptoms of Internal Derangement of the Knee Joint[1]
Symptoms of internal derangement of the knee joint are similar regardless of the cause.
Sudden mechanical interference with joint function in an other- [illegible]
Local pain, swelling and tenderness due to a resulting synovitis
Variation of the site of pain and swelling depending upon the causative lesion.
Recurrences with "trick knee" not infrequent.

Internal derangement of the knee joint results from lesions of the semilunar cartilages, injury to the cruciate and lateral ligaments, loose bodies, traumatic lesions of the synovial membrane and infrapatellar fat pad, injury of the tibial spine, and exostoses [18] *The causes of internal derangement of the knee joint have been classified as follows*

Semilunar cartilages (injuries or displacement).

Injuries to lateral ligaments (internal or external)

[illegible]

Cruciate ligaments.

Stretching or tearing of the anterior or posterior ligaments

Loose bodies.

Synovial origin (chronic synovitis, rheumatoid arthritis, osteochondromatosis).

Articular origin (osteochondritis dissecans, osteochondral fractures; detached osteophytes of degenerative joint disease).

Soft tissue.

Adhesions

Semilunar fat pads (*hypertrophic fringes*)

Synovial membrane—traumatic lesions.

Recurrent dislocation of the patella (slipping patella).

Fractures of the spine of the tibia

Exostoses.

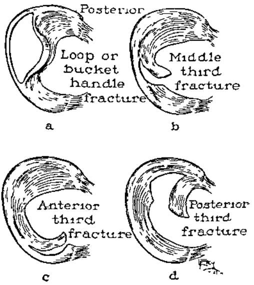


FIG. 202 —Types of injury to the semilunar cartilage (Henderson, courtesy of Surg, Gynec and Obst)

SEMILUNAR CARTILAGES

The semilunar cartilages are attached to the tibia and to the internal and external lateral ligaments[34] The external of these is smaller, more round and more movable than the internal, and is less likely to be "nipped" between the bones.[37] The fibrous semilunar cartilages are torn readily Some authors state that injuries or displacement of these structures are the most common causes of internal derangement of the knee joint.

In 300 instances of knee joint injury, Speed reported that the internal semilunar cartilage was damaged in 153 cases, the external cartilage only 25 times.[34] Mauck, however, states that the primary damage occurs ... crucial

28

nick-

icom-

of the femur upon a fixed tibia, with the knee flexed and abducted; in damage to *the external cartilage, the femur is rotated outward on the tibia with the knee* flexed and adducted, the foot firm on the ground. Injuries which damage the internal cartilage are much more common than those affecting the external. The latter is not attached to the lateral ligament and is more secure since it fits the anatomic contour of the joint more closely.

Symptoms of Tears or Dislocations of Semilunar Cartilage
[illegible] of the joint.
Sudden pain in the knee and leg, with *local tenderness* and *swelling*
Local site of tenderness. If anterior and mesial between the joint surfaces, anterior horn tears of the internal cartilage are suspected, if posterior and mesial, posterior horn tears of the internal cartilage, if externally, the external semilunar cartilage
Development of synovitis subsequent to the injury.

[illegible]

[illegible] fin[illegible] de[illegible] pir[illegible] chondritis dissecans. *Air arthography* has been used diagnostically in lesions of semilunar cartilages.[3,11,17,18,28,32]

Treatment of Lesions of the Semilunar Cartilage.—In acute cases, manipulation with or without anesthesia is advisable, some patients do not experience recurrences. Attempt to reduce the lesion immediately, before [illegible]

Ninety per cent of cases show marked improvement following operation, 35 per cent of "cures" can be expected with non-operative methods In general, the younger the patient, the quicker is the recovery.

Reduction of Tear of a Semilunar Cartilage.—With the patient lying on [illegible]

snap may be felt, the leg may be extended fully and the knee feels normal *to the patient.* A posterior molded splint is then worn until pain and swelling have subsided. Shands advises immobilization of the joint for at least *three weeks by rolls of cotton, thin board splints* and an elastic

muscle and massage are used to maintain muscle tone *Absorption of joint* fluid may be hastened by the use of infrared heat or diathermy.

two months following operation It has been stated that "There is no more danger in opening a knee joint than in opening an abdomen."[21] The *older concept that opening a knee joint resulted in ankylosis or stiffness is no longer valid Quadriceps setting exercises started the day of operation* and repeated regularly throughout convalescence shorten disability and *prevent muscle atrophy.*

SEMILUNAR CARTILAGE CYSTS

These are found in approximately 3 per cent of cases of internal derangement of the knee joint which come to operation[5] The commonest site of such cysts is anterior to the external lateral ligament of the knee joint in the middle of *the external semilunar cartilage, on the outer aspect of the* knee. These cysts are often multiple and contain a gelatinous material *Such cysts produce pain on walking, and especially in* climbing or

Other Lesions of the Semilunar Cartilages.—These include ossification (rare) and congenital variations in their size, shape and attachments.

The literature contains only 78 reported cases of calcification of the menisci of the knee joint. The calcium is usually laid down in parallel layers beginning at the periphery of the cartilage and involving the entire

length. The condition often produces no symptoms and may be discovered by accident or in others, symptoms may be severe enough to necessitate removal of the cartilage *One may see pain, swelling and limitation of motion.*

The calcification may be primary (non-traumatic) or secondary (following trauma). *In the primary cases, degenerative joint disease of the knees* usually is present.[35]

INJURIES TO THE LATERAL LIGAMENTS

The lateral ligaments, which are relatively feeble, are relaxed in flexion and tense in extension. **The internal ligament is more frequently strained than the external.**

partial

han the

internal

th pain,

gament

There is also an increased lateral mobility of the knee.

Treatment —Immobilization in plaster in complete extension for six weeks if complete tear occurs, in partial tears, immobilization for at least *three weeks.*[7] The splints may be removed daily for physical therapy, in severe injuries, motion of the joint is forbidden

When the patient becomes ambulatory, apply a knee brace to prevent hyperextension and excessive lateral motion and raise the heel on the affected side ½ inch to prevent ligamentous strain [13]

Badgeley[1] advises surgical repair in the case of complete tears, reconstruction may be accomplished with strips of fascia lata

Minor *tears may be treated by procaine injection and a supporting* bandage.

CRUCIATE LIGAMENT INJURY

The very *powerful crucial ligaments are more or less tense in any extreme* position of the joint One or both cruciate ligaments may be torn, rupture occurs only following severe trauma

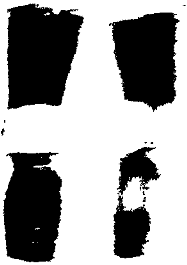
Severe *injuries of the knee joint often produce tear of a semilunar* cartilage and of a lateral ligament with rupture of the anterior cruciate ligament.

Symptoms.—1. Instability and marked *swelling of the knee joint* 2 Hyperextension and increased anterior displacement of the tibia (in the case of a torn anterior cruciate ligament) 3 Increased displacement *posteriorly (if the posterior cruciate ligament is torn).*

Treatment —Tears of both ligaments: plaster cast immobilization for two or three months (knee kept at about 15 degrees flexion)

If a stable knee is not produced, one may attempt to reconstruct the cruciate ligaments with fascia lata.[4]

The **Jones knee cage** has been used with success in the treatment of chronic relaxations of the crucial and lateral ligaments, with pseudolocking of the knee and excessive rotation and slipping of the tibia upon the femur.



LOOSE BODIES WITHIN THE KNEE JOINT

of lateral ligaments, 18 per cent, recurrent patellar dislocations, 10 per cent, cruciate ligament tears 4 per cent; semilunar cartilage cysts, 3 per cent, and ossified semilunar cartilage, 0 3 per cent.

Loose bodies are derivatives of synovial membrane or portions of articular

detached articular ecchondrosis; detached intra-articular epiphyses, loose bodies of *osteochondritis dissecans, osteochondromatosis and synovial* chondroma They are most common in degenerative joint disease They may be of fibrinous or cartilaginous origin

Osteocartilaginous bodies in joints should always be removed in young individuals unless contraindicated. They may cause any degree of joint symptoms and disability. Loose bodies are differentiated from semilunar

(including variability of location of the loose body).

OSTEOCHONDRITIS DISSECANS (*See* previous chapter)

TRAUMATIC OSTEOCHONDRITIS (CHONDROMALACIA) OF THE PATELLA

Most writers believe this condition results from acute trauma It is thought that the subchondral bone is compressed following a blow to the patella and areas of necrosis develop in the patella and femoral condyles If repair does not occur, the injured bone may be walled off and form a sequestrum Gross interference with nutrition of the overlying cartilage then may occur, resulting in degeneration of the cartilage, irritation and hypertrophy of the synovium with effusion and finally degenerative joint disease [ta]

Recently Bronitsky[6] described 29 cases (31 knees), of which 25 8 per cent denied any history of acute trauma The youngest patient in whom the diagnosis was verified at operation was thirteen years of age

Symptoms described by Bronitsky are those of chronic knee discomfort occurring *weeks or months after the post-traumatic symptoms have subsided* Pain is present especially on climbing hills or stairs, and on kneeling or squatting, patients complain also of morning stiffness and aching after sitting in one position. The leg feels weak and there is a tendency for the

[illegible] be repeated [illegible] with severe [illegible] at times followed by effusion into the joint. [illegible] under the patella on extending the knee [illegible] of [illegible] from a "click" to rough [illegible] "creaky knees" are believed due to this process.[20] [illegible] patella is pressed against the medial femoral [illegible] be thickened and tender. Synovial fluid [illegible] few cells and no evidence of infection [illegible]



Fig 293.—Jones knee [illegible] knee joint (osteochondromatosis). (Holmes [illegible])



Fig. 294

[illegible] physical therapy) should be given a [illegible] conservative therapy, partial chondrec[illegible] Thus far, however, insufficient time [illegible] of surgical treatment. Some [illegible][23]

[illegible] n young [illegible] full [illegible]

[illegible]

sional sharp pains and some limp when the knee joint is involved. In some, the cartilaginous bodies may be palpated in the suprapatellar space or popliteal area [39]

[illegible] recommended to [illegible] If the synovial [illegible] advised.

OSTEOCHONDRAL FRACTURES

These may occur in the patella, femoral condyles, etc , and require surgical removal if symptoms are produced.

RECURRENT DISLOCATION OF THE PATELLA

This may be complete or incomplete, the dislocation occurring laterally on active extension of the knee against force. When the lesion has been reduced, a tight compression bandage should be applied and the joint rested Symptoms are as follows (1) Most common in females (2) Dislocation is usually outward. (3) May produce extreme pain and disability (4) Patella returns to position in extension of the knee

If recurrences occur despite the use of braces, surgical treatment is indicated, with transference of the patellar insertion inward

FRACTURES OF THE TIBIAL SPINE

Fractures of the tibial spine are rare [39] Avulsion of either tubercle or the whole spine may occur. The outstanding symptom is the presence of a bony block, causing locking. There is also pain, tenderness and marked swelling of the joint; the patient cannot extend the knee In most cases, the fragment must be excised. In some cases with very little displacement of the fragment, one may immobilize the knee for four weeks, and provide support for the knee when the patient becomes ambulatory The fat pad behind the patellar tendon is occasionally pinched on extension of the knee.[35] If this trauma is repeated, hemorrhage, organization and calcification may occur in the fatty tissue

SOFT TISSUE LESIONS AFFECTING THE KNEE

Injury to the infrapatellar fat pad results in tenderness, pain on extension of the knee, pain on climbing stairs, stiffness, weakness and occasional locking of the knee. Ossification has been reported occasionally in the infrapatellar[39] bursa and fat pad. Treatment consists of elevating the heel of the affected side from ½ to 1 inch, support to the knee to prevent full

knee to buckle. There may be repeated momentary locking with severe pain lasting only a few seconds, at times followed by effusion into the joint.

Examination *reveals crepitus* under the patella on extending the knee from the flexed position All grades of crepitus from a "click" to rough grating are found. Many "squeaky knees" are believed due to this process[20] Pain is produced when the patella is pressed against the medial femoral condyle. The synovium may be thickened and tender. Synovial fluid *when present is clear* and shows few cells and no evidence of infection. Roentgenograms are negative.



FIG. 295 —Multiple calcified bodies in the knee joint (osteochondromatosis) (Holmes and Robbins)

Conservative treatment (rest and physical therapy) should be given a trial In cases not responding to conservative therapy, partial chondrectomy or patellectomy is advised.[6,12] Thus far, however, insufficient time has elapsed to permit proper evaluation of surgical treatment. Some disapprove particularly of patellectomy[2,23]

OSTEOCHONDROMATOSIS

marbles.

Osteochondromatosis is relatively uncommon; multiple intra-articular [illegible]

Symptoms are usually those complained of in arthritic joints, with occasional sharp pains and some limp when the knee joint is involved In some, the cartilaginous bodies may be palpated in the suprapatellar space or popliteal area.[29]

[illegible] recommended to [illegible] If the synovial [illegible] advised

OSTEOCHONDRAL FRACTURES

These may occur in the patella, femoral condyles, etc , and require surgical removal if symptoms are produced

RECURRENT DISLOCATION OF THE PATELLA

This may be complete or incomplete, the dislocation occurring laterally on active extension of the knee against force When the lesion has been reduced, a tight compression bandage should be applied and the joint rested. Symptoms are as follows (1) Most common in females (2) Dislocation is usually outward. (3) May produce extreme pain and disability (4) Patella returns to position in extension of the knee

If recurrences occur despite the use of braces, surgical treatment is indicated, with transference of the patellar insertion inward

FRACTURES OF THE TIBIAL SPINE

Fractures of the tibial spine are rare [30] Avulsion of either tubercle or the whole spine may occur The outstanding symptom is the presence of a bony block, causing locking There is also pain, tenderness and marked swelling of the joint, the patient cannot extend the knee In most cases, the fragment must be excised In some cases with very little displacement of the fragment, one may immobilize the knee for four weeks, and provide support for the knee when the patient becomes ambulatory The fat pad behind the patellar tendon is occasionally pinched on extension of the knee.[15] If this trauma is repeated, hemorrhage, organization and calcification may occur in the fatty tissue

SOFT TISSUE LESIONS AFFECTING THE KNEE

Injury to the infrapatellar fat pad results in tenderness, pain on extension of the knee, pain on climbing stairs, stiffness, weakness and occasional locking of the knee Ossification has been reported occasionally in the infrapatellar[29] bursa and fat pad. Treatment consists of elevating the heel of the affected side from ½ to 1 inch, support to the knee to prevent full

extension, massage and exercise of the surrounding muscles, and if necessary, surgical removal of the fat pad. Procaine injections may give marked symptomatic relief

eff

to

M

cartilaginous lesions developing on one or both of these bones. Occasionally, aborescent lipomata (resulting in severe changes in the articular surface of the patella and underlying femur), anterior bulge of hypertrophic fat and femoral-tibial intrusions without articular surface lesions were causative factors.

Lesions of the suprapatellar plica may produce mechanical disability of the knee simulating cartilage disability. This structure is a fold of synovia which represents a communication between the bursa and the joint and is especially prominent at the medial border. Pipkin[26] reports a series of 247 pneumo-arthrograms which demonstrate abnormalities of this fold. He states that such a plica exists in 78 per cent of adult knees, representing fusion of the suprapetellar bursa with the knee joint. In cases of open communication, fibrosis, hyalinization and calcification of the plica may occur and result in mechanical interference with the extensor apparatus. This produces a syndrome of aching, "popping" (pseudo-locking) and buckling of the knee, often with chronic effusion. In pneumo-arthrograms, the density of the plica serves as an index of synovial reaction and is of value in prognosis.

BIBLIOGRAPHY

1 Badgeley, C R Nelson's Loose Leaf Living Surgery, New York, Thomas Nelson & Sons, **3**, 180 L, 1935
2. Bauer, W Ann Rheum Dis, **9**, 402, 1950
3 Blonek, F, and Wolf, J Jour Iowa State Med Soc, **34**, 354, 1944
4 Bosworth, D. M, and Bosworth, B M. Jour Bone and Joint Surg, **18**, 178, 1936
5 Bristow, W R Jour Bone and Joint Surg, **17**, 605, 1935
6 Bronitsky, J · Jour Bone and Joint Surg, **29**, 931, 1947
7 Burns, B H, and Ellis, V H Recent Advances in Orthopedic Surgery, Philadelphia, The Blakiston Company, 1937.
8 Campbell, W C Surg Gynec, and Obst, **52**, 568, 1931
9 Colonna, P C · Surg, Gynec, and Obst, **53**, 698, 1921
10 Cox, F J Surgery, **17**, 93, 1945
11. Cullen, O. H, and Chance, G Q Brit Jour Surg, **30**, 241, 1943
12 Ensign, D C, and Sigler, J M Ann Rheum Dis, **9**, 402, 1950
13 Evans, W A, Jr Am Jour Roentgenol, **43**, 860, 1940
14 Fairbank, H A T Proc Roy. Soc Med, **30**, 427, 1936
15 Fisher, A. G T Internal Derangements of the Knee Joint, 2d ed, New York, The Macmillan Company, 1933
16 Ghormley, R K, and Dockerty, M B · Jour Bone and Joint Surg, **25**, 306, 1943
17. Harmon, P H · Jour Bone and Joint Surg, **26**, 838, 1944
18 Hauch, P P Brit Jour Radiol, **17**, 70, 1944
19 Hauser, E D W Jour Bone and Joint Surg, **24**, 307, 1942
20 Hench, P S, *et al* Ann Int Med, **28**, 324, 1948
21 Henderson, M S Surg Clin North America, **17**, 1207, 1937
22 Kuhns, J G Ann Rheum. Dis, **9**, 402, 1950
23 Jones, H T Jour Bone and Joint Surg, **6**, 407, 1924
24 Leibman, C, and Iseman, R M Am Jour Roentgenol, **43**, 865, 1940.
25 Levinthal, D H · Surg Clin North America, **23**, 181, 1943

C V Mosby Company, 1937
32 SOMERVILLE, E W · Proc Roy Soc. Med , 36, 663, 1943
33 STACK, J K , and CHASTEN, S Am Jour Surg , 78, 570, 1949
34 SPEED, K Wisconsin Med Jour , 40, 1051, 1941
35 STEINDLER, A · Jour Am Med Assn , 84, 16, 1925
36 TERHUNE, S R , EDDLEMAN, T S , THOMPSON, S B , and READ, B S Jour Bone and Joint Surg , 25, 663, 1943
37 TREVES, SIR F : Surgical Applied Anatomy, 11th ed , London, Cassell & Co , Ltd , 1948
38 WEAVER, J B Jour Bone and Joint Surg , 24, 873, 1942
39 WILMOTH, C L · Jour Bone and Joint Surg , 23, 367, 1941

CHAPTER 54

THE PAINFUL SHOULDER

By Wallace Graham, M.D.

Excluding acute traumatic lesions such as fractures, dislocations, contusions, sprains and ruptured tendons, 85 to 90 per cent of painful shoulders arise from calcareous tendinitis, acute and chronic bursitis (with adhe-

dary to calcification in the short rotator tendons, particularly the supraspinatus.

Arthritis involving the shoulder joint alone causes not more than 5 per cent of all cases of painful shoulders Of the 140 or more bursæ in the body (33 in each upper and 37 in each lower extremity), the subdeltoid is the one which most often gives symptoms.

Steindler classified the lesions about the shoulder joint into bursal lesions, primary injuries to tendons or the musculotendinous cuff overlying

joint capsule, acromioclavicular

ng baseball pitchers, orchestra conductors, violinists and those who use their arms vigorously; the terms "glass arm" and "musician's shoulder" are well-known in the baseball and musical professions.

In any pain about the shoulder joint, the physician should determine if this is traumatic, acute or chronic, and whether the main offender is

areas.

be seated on a

parison is made

elling, muscular

greater tuberosity or

patient is then asked to slowly raise both arms laterally and to fully extend them above the head. Another useful test is to have the individual place his involved forearm horizontally behind his back (without assistance from the non-involved arm)

In the general examination, attention must also be directed to the cervical spine, the chest, and diaphragmatic region as disease in these areas may cause referred shoulder pain.

Full elevation of the arm includes rotation of the humerus upward on the scapula, and of the scapula on the chest wall; when the arm is fully elevated, the glenoid faces upward rather than outward[47] The greater

tuberosity of the humerus slides under the acromion during elevation of the arm, requiring a normal subdeltoid bursa for smooth, painless performance. If dense bursal adhesions or fusion of the shoulder joint are present, the arm can be elevated almost horizontally due entirely to rota-

contributed by synchronous participation of the sternoclavicular, acromio-

is completed by motion of scapula on the chest wall, is incorrect." On the contrary, motion occurs simultaneously in all joints of the region, each contributing its share. During early phases of elevation of the arm, the sternoclavicular joint passes through its greatest range of motion, in the terminal phase, the acromioclavicular. "At the glenohumeral and scapulo-

the termina-

of elevation

Ankylosis of any of the joints causes a permanent loss in degree of movement in direct proportion to the amount of movement that joint should have contributed.[43]

Approximate Frequency of Causes of a Painful Shoulder
Subdeltoid or subacromial bursitis (associated most often with *calcareous deposits*, particularly *in the supraspinatus tendon*)—at least 80 per cent of all cases.
Myofibrositis—8 per cent
Arthritis of the shoulder joint—5 per cent
Rupture of the supraspinatus tendon (common following industrial accidents to the shoulder)
Neuritis—a very infrequent cause of pain in the shoulder
Other causes *Cervical rib and scalenus anticus syndrome, arthritis of the cervical spine*, with narrowing of the intervertebral foramina (disco-

SUBDELTOID (SUBACROMIAL) BURSITIS

Numerous synonyms for this condition include: *periarthritis of the shoulder, subacromial bursitis*, painful shoulder, calcifications of the supraspinatus tendon, periarthritis humeroscapularis, calcified bursitis, bursitis calcarea subdeltoidea or subacromialis, Duplay's disease, etc.[22, 53]

Codman, who has been the pioneer in this field, believes that bursitis, like peritonitis, always has a cause and that many of the lesions producing painful symptoms in the shoulder are *due primarily to involvement of the supraspinatus tendon* with secondary lesions of the bursa.[14] Mosley[53] has stressed the fact that different lesions may give rise to similar symptoms and signs, and that "periarthritis" of Duplay[22] should be differentiated into: (a) calcified deposits in the rotator cuff, (b) ruptures of the rotator cuff, (c) lesions of bicipital tendons, (d) secondary bursitis, or (e) stiff, or frozen, shoulder.

Causes of Subdeltoid Bursitis· 1. *Trauma* (overuse; prolonged hyperabduction, rupture of the supraspinatus, infraspinatus or long head of the biceps; acromioclavicular dislocation; fracture of the greater tuberosity; direct injury to the top of the anterior part of the shoulder with the arm in dorsal flexion; irritation from osteophytes).

2. Subdeltoid bursitis with adhesions occurs not infrequently in older patients who are confined to bed for long periods (as with coronary artery occlusion), or in association with diseases of the nervous system. The roughened bursal surfaces probably become adherent during this time (because of lack of motion), also, when confined to bed the shoulders are often used as weight bearing joints.

Calcifications may occur in the injured areas of the tendon, with secondary bursal involvement.

3. The supraspinatus tendon is so situated that it is subject to frequent mild trauma, being pinched between the acromion and the head of the humerus.[20]

Calcification in the supraspinatus tendon has not been reported among children.

4. Moseley[53] feels that an area of ischemia from chronic postural strain [illegible]

lesions in contiguous tissues, such as the supraspinatus tendon, the floor of the bursa or the musculotendinous cuff composed of the tendons of the supraspinatus and infraspinatus, teres minor, and subscapularis muscles.

Bosworth found *2.7 per cent of 6,061 unselected persons to have calcium deposits about the shoulder.* The majority of these developed slowly over a period of months

[illegible] been demonstrated in many other regions,

olved.

ulder.

Trauma may [illegible]
tional maneu[illegible]
abducted for [illegible]
individuals, especially those who have done laboring work for many [illegible] there is found a degeneration of the tendons of the short rotator muscles of the shoulder; under such conditions, mild trauma may produce injury or rupture of these tendons. In a series of 52 autopsies in an institution for elderly individuals, shoulder specimens revealed that some extent of rupture of the fibers of the supraspinatus tendon had occurred during the lives of 39 per cent of these patients[8] Complete ruptures cause far more

serious symptoms than partial ruptures and often lead to permanent disability.

Injuries to the Shoulder and "Painful Shoulder."—Following trauma to the shoulder, as in fractures, the wall of the subdeltoid bursa may be torn and hemorrhage may occur into it; in injuries or dislocations in the acromioclavicular region, the bursa may be distended with fluid as a result of an exudative reaction.[39] This reaction may be mild with the production of only a slight exudate and congestion, but with the formation of adhesions between the layers of the bursa Adhesions are produced within the bursa, and one may find a villous synovitis (from hypertrophy of the synovial folds), or fatty degeneration of these villi The result of such pathologic changes is decreased motion of the shoulder joint

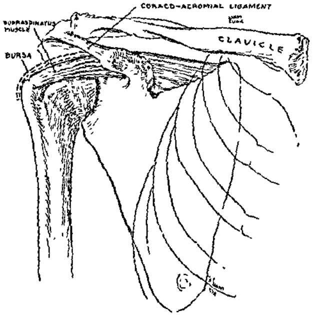


FIG 206 —Drawing showing relation of subacromial bursa to supraspinatus tendon coraco-acromial ligament and greater tuberosity of the humerus (Haggart and Allen, courtesy of Surg Clin. North America)

The origins and insertions of muscles around the shoulder joint may be damaged by straining or twisting One occasionally sees a **traumatic tendinitis of the long head of the biceps** producing localized pain anteriorly over the shoulder, pain on raising the arm and tenderness over the bicipital groove. Another uncommon lesion is *traumatic myositis of the scalenus anticus muscle*, causing pain at the shoulder radiating down the arm, tenderness over the scalenus anticus muscle mass (behind the lower portion of the sterno-mastoid) and some restriction of motion Both of these conditions may be dramatically relieved by procaine infiltration[48]

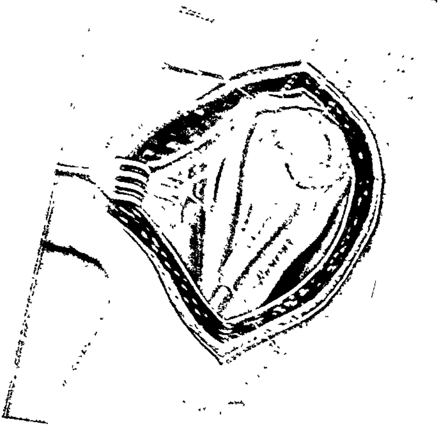
... include dislocation of ...

... of the supraspinatus tend...

... or inflammatory chang...

... supraspinatus tendon which ofte...

... complete ruptures do not ...



calcific deposits into bursæ, but to the increased tension produced in the tendons, when the deposit ruptures into the bursa "immediate relief" occurs [42]

Symptoms of Acute Subdeltoid Bursitis (Periarthritis of the Shoulder)
Agonizing *pain in the region of the shoulder joint*, aggravated by motion and *worse at night* (often preventing sleep).
Radiation of the pain at times into the neck, arm, insertion of the deltoid, forearm and even to the finger tips
Limitation of motion (because of pain), *extreme pain on even the slightest abduction or rotation in acute cases*
Tenderness in the region of the greater tuberosity of the humerus just below the acromial process (and at times over the entire shoulder), the coracoid process and bicipital groove may also be sensitive to pressure
Roentgen findings (in about 50 per cent of cases) [71]

Location of the Subdeltoid Bursa —*The subdeltoid, subacromial and subcoracoid bursæ are probably one structure, although these may be separated by thin films of tissue, this is the largest bursa in the body* The bursa lies over the greater tuberosity of the humerus and extends downward under the upper portion of the deltoid muscle between its tendon and the tendon of insertion of the supraspinatus and the head of the humerus It extends under the acromion and the coracoid and under the origin of the short head of the biceps It may be as large as the palm of the hand It is lined with a thin synovial membrane, the small amount of fluid present allows movement without friction between the two surfaces *The subdeltoid bursa is intimately connected with freedom of motion in the shoulder joint* [89]

Symptoms of Subdeltoid Bursitis.—The *patient with acute subdeltoid bursitis* develops a pain usually several inches above the insertion of the deltoid muscle. This may be aching or severe and associated with sharp stabbing pain on certain motions, especially that of abduction, in acute cases, this is often agonizing There may be a history of trauma **The outstanding symptom is severe pain in the shoulder**

Not infrequently, the onset is sudden The patient holds the arm to the side of the body, supported by the good arm *One of the misleading points concerning this pain is that it may be referred down the lateral aspect of the arm to the elbow, and even into the forearm and hand* The pain is often so severe that the patient is willing to undertake any procedure for its relief

In acute cases, exquisite *tenderness* is noted in the region of the greater tuberosity of the humerus If the examiner can gain the patient's confidence, assuring him that he will undertake no sudden painful motions of the arm, he will be able to move the shoulder joint to some extent antero-posteriorly without discomfort to the patient, however, any attempt at abduction may cause excruciating pain

Two procedures are useful in diagnosis of this condition (1) if the arm is abducted no pain is experienced through the first 15 degrees of motion, however, if the abduction is continued, the patient will experience slight or severe pain; (2) the patient is unable (because of pain, and later, of adhesions) to place the arm on the affected side horizontally behind the back in an attempt to grasp the opposite elbow When this latter motion

Other traumatic lesions about the shoulder include dislocation of the ... he supraspinatus tendon

... *of inflammatory changes within the subdeltoid bursa are tears in the supraspinatus tendon, which often result in calcified deposits within this tendon.* Complete ruptures do not cause calcified deposits



FIG 297 —The subdeltoid bursa (Baer)

Others state that the lesion begins as a tendinitis[12] *which later undergoes* local necrosis and calcification or that the constant movement of the soft parts on each other leads to fraying, roughening or obliteration of the smooth inner surface of the bursa.[56]

According to most writers the degenerative changes and calcium deposits in avascular tendons are symptomless and inflammation occurs only when the calcium ruptures into the vascular peritendinous tissues.[37] In contrast Howorth believes the acute attacks are not due to rupture of tendinous

varying in size from a few millimeters to several centimeters in diameter

During the acute stage, the bursa may be greatly distended with fluid

In cases of indeterminate shoulder pain, a view of the base of the neck should be made to demonstrate the presence or absence of cervical rib. Lateral and oblique views of the cervical spine will indicate the probability of referred pain from disc lesions or cervical osteoarthritis changes causing nerve root encroachment [84]

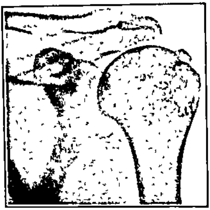


FIG 299 —Acute subacromial bursitis Note large mass of calcified deposit Sharply localized pronounced bone atrophy is indicated by arrows (Haggart and Allen, courtesy of Surg Clin North America)

In taking such a roentgenogram in an effort to locate a calcified bursa, pictures should be taken in two positions, one with slight internal rotation of the humerus and the other with slight external rotation [84] In some cases, the calcified deposits may not be visualized by roentgen-ray if there is complete internal rotation of the arm. Roentgenograms of both shoulders should be taken for comparison as bilateral calcified bursæ may be found, one of these may be absolutely painless. Although roentgen changes are often present, *negative roentgen-ray findings do not rule out subdeltoid bursitis* On the other hand, calcified deposits may be found in as many as 25 per cent of shoulders with no pain

Bosworth found calcium in routine fluoroscopic examination in one or both shoulders in 2 7 per cent of 6061 supposedly normal persons of the white collar class [10] These were found most often in males in the period of greatest activity of adult life The formation or regression of the deposits did not seem to be influenced by serious illness, arthritis or local or systemic infection Occupations requiring constant prolonged abduction of the arms fostered the formation of calcium deposits in the shoulder A single trauma *per se* did not cause calcium deposition *Fluoroscopically visible calcium deposits formed within as short a period as two months*, but most deposits required a longer time Small and medium sized deposits dis-

is attempted by the patient, he invariably tries to assist the affected arm by the hand of the sound arm. *In most acute instances, pain is so severe that sleep is impossible*; in many, large doses of morphine do not completely relieve the discomfort

Kaplan has emphasized the [illegible]
ing [illegible]
Such [illegible]
over [illegible] shoulder, radiating to the insertion of the deltoid or to the elbow, wrist or fingers. The distribution of the pain is not a reliable guide in locating the lesion.

During the *acute stage of the process* there may be a moderate leukocytosis and a slight increase in the sedimentation rate.



FIG 298 —Calcification of tendon and bursa—preoperative (*A*); (*B*) postoperative excellent result of operative treatment (Rubert, courtesy of Arch. Surg)

Mosley[54] points out that "as the acute phase runs its limited course, the clinical picture mirrors the process in the deposit." His findings at operation show that the deposit in the acute phase is semi-liquid, like tooth paste, or milky and under great tension. In the silent or chronic phase it is in the state of a dry powder The attack may subside slowly by reduction of the water content of the deposit, and probably by resorption of some or all of the calcium. The pain may also rapidly disappear by rupture of the deposit into the bursa or along the fascial planes. Acute pain may be relieved by this sudden reduction in tension, but if the deposit is large, a severe inflammatory reaction may follow. This may be a frequent cause of the more chronic stiff and painful shoulder.[53]

Roentgen Findings.—Fractures or dislocations must be ruled out by careful roentgen examination if there has been a history of trauma. A complete history, physical examination, and roentgen examination of this region (and sometimes of the cervical spine) will usually reveal the diagnosis

Roentgen examination of the shoulder region in cases of subdeltoid bursitis reveals (in about 50 per cent of the patients) a calcified deposit over the greater tuberosity and an associated bony atrophy in this region (if the deposit has been present for some time).[25]

On roentgen examination, shadows of varying intensity are cast by the calcareous deposits; these may be thin and hazy or as dense as bone The deposits may be single or multiple and may be unilateral or bilateral,

phosphate and carbonate; traces of iron, magnesium and chlorides have also been found.

Treatment for Acute Traumatic Subdeltoid Bursitis.—Acute traumatic bursitis is often associated with injuries to other structures of the shoulder, those most commonly affected include the acromioclavicular joint, fractures of the humerus (head, neck or tuberosities), dislocation of the shoulder and damage to the supraspinatus and infraspinatus tendons. In these cases, attention must be given not only to the "bursitis" but also to the associated pathology. In other cases, the trauma may be slight, such as excessive use of the arm in abduction, twists and wrenches of the shoulder joint, blows upon the shoulder, or falls upon the hand or arm with the arm in partial abduction

Most patients with acute traumatic subdeltoid bursitis respond well to

in order to avoid adhesions

Kaplan and others[47] have reported good results in these patients by *aspiration of the bursa, injection of 5 to 10 cc. of 1 per cent procaine, followed by a thick axillary pad with a sling and swathe dressing in order to maintain a moderate amount of abduction* An ice bag may be applied if this affords relief in the acute stage. Active exercises are begun in three to five days

In a recent report, Hollander, *et al*[40] suggest that the injection of 25 mg of hydrocortisone acetate suspension into the inflamed bursa can quickly suppress the inflammation, apparently without recurrence The technic for injection is similar to that for procaine injection into the bursa, or to that for hydrocortisone injection into joints This treatment has been found effective in acute bursitis, whether or not calcium was present Other workers have administered ACTH or cortisone systemically with benefit, but an acute localized and self-limited condition would hardly justify the employment of such potent hormones systemically (*See also* Chapter 17)

Treatment for Acute Subdeltoid Bursitis When Calcification is Present —*The choice of therapy* in this extremely common condition *seemed to depend in the past in large part upon the field of specialization of the writers on this subject*, thus, the roentgenologist claimed excellent results with roentgen-ray therapy; the physical therapist stated that diathermy, galvanism or iontophoresis were the treatments of choice, the surgeon preferred surgical incision and removal of the calcified material; the orthopedist suggested a regimen including splints and traction, while medical men (and some surgeons) found that good results followed procaine injec-

and speed of action of hydrocortisone injection may supplant many other measures at least in treating *acute* subdeltoid bursitis

Calcium deposition in tendons ...

large the de[illegible]

hyperemia.

appeared in some cases without symptoms but *large deposits sooner or later resulted in a painful shoulder, although they remained quiescent and symptomless for months or years*

Roentgenologically, the calcified area must be differentiated from the anterior tip of the acromion, chip fractures of the tuberosities, atrophy of the proximal end of the shaft with subchondral cysts (sometimes seen in arthritis), primary or metastatic malignancy, leukemia, Hodgkin's disease and free bodies in the joint.

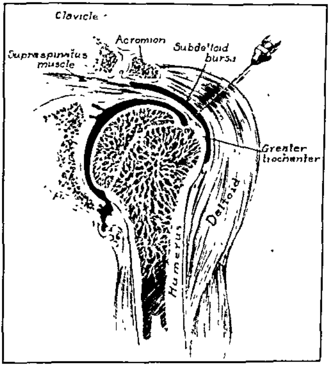


FIG 300 —Injection of subdeltoid bursa. (Haldeman and Soto-Hall, courtesy of Jour Am Med Assn)

Causes of Calcification in Patients With Subdeltoid Bursitis.—*The calcified deposits actually occur in the tendons of the short rotator muscles,* most commonly in the supraspinatus tendon, but also sometimes in the tendons of the subscapularis, teres minor and infraspinatus.[26] Most observers feel that *deposition of calcium salts occurs in areas of local necrosis in the tendon fibers.* Repeated minor trauma in these regions probably results in localized death of tissue Because of the poor circulation, these necrotic areas become calcified

The Nature of the Calcium Deposits.—The deposits may be unilateral or bilateral, single or multiple Such deposits may vary in consistency from a milky fluid or chalky material or to discrete calcified nodules or massive calcification. This material is usually the consistency and color of white tooth paste. The areas of calcification are composed of calcium

phosphate and carbonate; traces of iron, magnesium and chlorides have also been found.

Treatment for Acute Traumatic Subdeltoid Bursitis.—Acute traumatic bursitis is often associated with injuries to other structures of the shoulder; those most commonly affected include the acromioclavicular joint, fractures of the humerus (head, neck or tuberosities), dislocation of the shoulder and damage to the supraspinatus and infraspinatus tendons In these cases, attention must be given not only to the "bursitis" but also to the associated pathology. In other cases, the trauma may be slight, such as excessive use of the arm in abduction, twists and wrenches of the shoulder joint, blows upon the shoulder, or falls upon the hand or arm with the arm in partial abduction

Most patients with acute traumatic subdeltoid bursitis respond well to

in order to avoid adhesions

Kaplan and others[47] have reported good results in these patients by *aspiration of the bursa, injection of 5 to 10 cc of 1 per cent procaine, followed by a thick axillary pad with a sling and swathe dressing in order to maintain a moderate amount of abduction.* An ice bag may be applied if this affords relief in the acute stage Active exercises are begun in three to five days

In a recent report, Hollander, *et al*[40] suggest that the injection of 25 mg. of hydrocortisone acetate suspension into the inflamed bursa can quickly suppress the inflammation, apparently without recurrence The technic for injection is similar to that for procaine injection into the bursa, or to that for hydrocortisone injection into joints This treatment has been found effective *in acute bursitis, whether or not calcium was present* Other workers have administered ACTH or cortisone systemically with benefit, but an acute localized and self-limited condition would hardly justify the employment of such potent hormones systemically. (*See also* Chapter 17)

Treatment for Acute Subdeltoid Bursitis When Calcification is Present. —*The choice of therapy* in this extremely common condition *seemed to depend in the past in large part upon the field of specialization of the writers on this subject*; thus, the roentgenologist claimed excellent results with roentgen-ray therapy, the physical therapist stated that diathermy, galvanism or iontophoresis were the treatments of choice, the surgeon preferred surgical incision and removal of the calcified material, the orthopedist suggested a regimen including splints and traction, while medical men (and some surgeons) found that good results followed procaine injection and washing out the bursa by needle irrigation, or multiple punctures in the wall of the bursa. The variety of treatments recommended strongly suggests that none are specific Hollander[40] suggests that the simplicity and speed of action of hydrocortisone injection may supplant many other measures at least in treating *acute* subdeltoid bursitis

Calcium deposition in tendons is a reversible process, unless they are large the deposits are generally absorbed by any process involving acute hyperemia. Many deposits disappear rather rapidly after almost any or no

appeared in some cases without symptoms but *large deposits sooner or later resulted in a painful shoulder, although they remained quiescent and symptomless for months or years.*

Roentgenologically, the calcified area must be differentiated from the anterior tip of the acromion, chip fractures of the tuberosities, atrophy of the proximal end of the shaft with subchondral cysts (sometimes seen in arthritis), primary or metastatic malignancy, leukemia, Hodgkin's disease and free bodies in the joint.

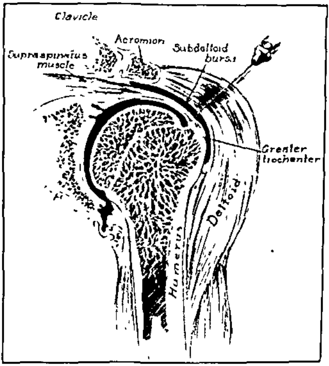


FIG 300 —Injection of subdeltoid bursa (Haldeman and Soto-Hall, courtesy of Jour Am Med Assn)

Causes of Calcification in Patients With Subdeltoid Bursitis.—*The calcified deposits actually occur in the tendons of the short rotator muscles,* most commonly in the supraspinatus tendon, but also sometimes in the tendons of the subscapularis, teres minor and infraspinatus [28] Most observers feel that *deposition of calcium salts occurs in areas of local necrosis in the tendon fibers.* Repeated minor trauma in these regions probably results in localized death of tissue. Because of the poor circulation, these necrotic areas become calcified

The Nature of the Calcium Deposits.—The deposits may be unilateral or bilateral, single or multiple. Such deposits may vary in consistency from a milky fluid or chalky material or to discrete calcified nodules or massive calcification. This material is usually the consistency and color of tooth paste. The areas of calcification are composed of calcium

phosphate and carbonate; traces of iron, magnesium and chlorides have also been found.

Treatment for Acute Traumatic Subdeltoid Bursitis.—Acute traumatic bursitis is often associated with injuries to other structures of the shoulder, those most commonly affected include the acromioclavicular joint, fractures of the humerus (head, neck or tuberosities), dislocation of the shoulder and damage to the supraspinatus and infraspinatus tendons. In these cases, attention must be given not only to the "bursitis" but also to the associated pathology. In other cases, the trauma may be slight, such as excessive use of the arm in abduction, twists and wrenches of the shoulder joint, blows upon the shoulder, or falls upon the hand or arm with the arm in partial abduction

Most patients with acute traumatic subdeltoid bursitis respond well to conservative therapy including temporary use of a sling, procaine injection into the bursa and analgesics Following the subsidence of pain, graduated exercises must be employed to prevent the formation of dense adhesions. In order to avoid adhesions

Kaplan and others[47] have reported good results in these patients by *aspiration of the bursa, injection of 5 to 10 cc. of 1 per cent procaine, followed by a thick axillary pad with a sling and swathe dressing in order to maintain a moderate amount of abduction* An ice bag may be applied if this affords relief in the acute stage Active exercises are begun in three to five days

In a recent report, Hollander, *et al*[40] suggest that the injection of 25 mg. of hydrocortisone acetate suspension into the inflamed bursa can quickly suppress the inflammation, apparently without recurrence The technic for injection is similar to that for procaine injection into the bursa, or to that for hydrocortisone injection into joints This treatment has been found effective in acute bursitis, whether or not calcium was present Other workers have administered ACTH or cortisone systemically with benefit, but an acute localized and self-limited condition would hardly justify the employment of such potent hormones systemically (*See also* Chapter 17)

Treatment for Acute Subdeltoid Bursitis When Calcification is Present. —*The choice of therapy* in this extremely common condition *seemed to depend in the past in large part upon the field of specialization of the writers on this subject*, thus, the roentgenologist claimed excellent results with roentgen-ray therapy, the physical therapist stated that diathermy, galvanism or iontophoresis were the treatments of choice, the surgeon preferred surgical incision and removal of the calcified material; the orthopedist suggested a regimen including splints and traction, while medical men (and

and speed of action of hydrocortisone injection may supplant many other measures at least in treating *acute* subdeltoid bursitis

Calcium deposition in tendons is a reversible process, unless they are large the deposits are generally absorbed by any process involving acute hyperemia Many deposits disappear rather rapidly after almost any or no

appeared in some cases without symptoms but *large deposits sooner or later resulted in a painful shoulder, although they remained quiescent and symptomless for months or years.*

[illegible]

and free bodies in the joint.

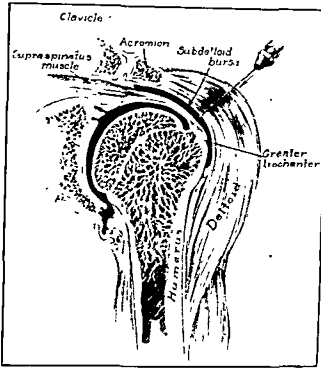


FIG 300.—Injection of subdeltoid bursa. (Haldeman and Soto-Hall, courtesy of Jour Am Med Assn.)

Causes of Calcification in Patients With Subdeltoid Bursitis.—*The calcified deposits actually occur in the tendons of the short rotator muscles,* most commonly in the supraspinatus tendon, but also sometimes in the tendons of the subscapularis, teres minor and infraspinatus.[26] Most observers feel that *deposition of calcium salts occurs in areas of local necrosis in the tendon fibers.* Repeated minor trauma in these regions probably results in localized death of tissue. Because of the poor circulation, these necrotic areas become calcified

The Nature of the Calcium Deposits.—The deposits may be unilateral or bilateral, single or multiple. Such deposits may vary in consistency from a milky fluid or chalky material or to discrete calcified nodules or massive calcification This material is usually the consistency and color of white tooth paste. The areas of calcification are composed of calcium

phosphate and carbonate; traces of iron, magnesium and chlorides have also been found.

Treatment for Acute Traumatic Subdeltoid Bursitis.—Acute traumatic bursitis is often associated with injuries to other structures of the shoulder, those most commonly affected include the acromioclavicular joint, fractures of the humerus (head, neck or tuberosities), dislocation of the shoulder and damage to the supraspinatus and infraspinatus tendons. In these cases, attention must be given not only to the "bursitis" but also to the associated pathology. In other cases, the trauma may be slight, such as excessive use of the arm in abduction, twists and wrenches of the shoulder joint, *blows upon the shoulder, or falls upon the hand or arm with the* arm in partial abduction

Most patients with acute traumatic subdeltoid bursitis respond well to conservative therapy including temporary use of a sling, procaine injection into the bursa and analgesics. Following the subsidence of pain, graduated exercises must be employed to prevent the formation of dense adhesions in order to avoid adhesions

Kaplan and others[47] have reported good results in these patients by *aspiration of the bursa, injection of 5 to 10 cc of 1 per cent procaine, followed by a thick axillary pad with a sling and swathe dressing in order to maintain a moderate amount of abduction* An ice bag may be applied if this affords relief in the acute stage Active exercises are begun in three to five days

In a recent report, Hollander, *et al*[40] suggest that the injection of 25 mg. of hydrocortisone acetate suspension into the inflamed bursa can quickly suppress the inflammation, apparently without recurrence The technic for injection is similar to that for procaine injection into the bursa, or to that for hydrocortisone injection into joints This treatment has been found effective in acute bursitis, whether or not calcium was present Other workers have administered ACTH or cortisone systemically with benefit, but an acute localized and self-limited condition would hardly justify the employment of such potent hormones systemically (*See also* Chapter 17)

Treatment for Acute Subdeltoid Bursitis When Calcification is Present. —*The choice of therapy* in this extremely common condition *seemed to depend in the past in large part upon the field of specialization of the writers on this subject*, thus, the roentgenologist claimed excellent results with roentgen-ray therapy, the physical therapist stated that diathermy, galvanism or iontophoresis were the treatments of choice, the surgeon preferred surgical incision and removal of the calcified material; the orthopedist suggested a regimen including splints and traction, while medical men (and some surgeons) found that good results followed procaine injection and washing out the bursa by needle irrigation, or multiple punctures in the wall of the bursa The variety of treatments recommended strongly suggests that none are specific Hollander[40] suggests that the simplicity and speed of action of hydrocortisone injection may supplant many other measures at least in treating *acute* subdeltoid bursitis

Calcium deposition in tendons is a reversible

treatment. Conservative measures are therefore recommended in most acute cases [37]

In these patients with acute subdeltoid bursitis with calcification, some attempt to *wash out the calcified material* after preliminary procaine injection It is often possible, by the use of two large gauge needles, to wash much of the soft calcified material out of these bursæ In addition to this, *roentgen therapy* may be employed and temporary splinting of the area in abduction for several days until the acute pain has subsided. Analgesics are required (aspirin and codeine or morphine). After several days, *passive and then active motion* is encouraged to prevent the development of a frozen shoulder.

The presence or absence of calcification is not *per se* of any significance in determining the response to irradiation. We have seen excellent results in these cases following roentgen therapy.

about six treatments are given, and if definite improvement does not occur during the next few weeks, no further roentgen therapy should be given. The pain may be aggravated for twenty-four hours after a treatment. (*See also* Chapter 6—Radiology—for Technic of Therapy)

Technic of Irrigation in Acute Bursitis.—Darrach and Patterson have claimed that patients with subdeltoid bursitis are returned to work much earlier than with any other method employed (after less than five days of disability in acute cases) [18] The materials required are· (1) two 18 gauge sterile needles ($2\frac{1}{2}$ inches long), (2) one 20 cc syringe; (3) 60 cc of a sterile 1 per cent solution of procaine; (4) one 2 cc. sterile hypodermic ... (5) 100 cc of sterile physiologic saline,

ine position,
l the region
large needles
m tenderness
rizontal line

as the coracoid process of the scapula) The *second* is about ½ inch behind ... on a level with the superior facet.

ates
shed

through the skin incision The point of the needle is directed ... orly

reached a depth of from ... needle within the bursa *The pressure within the bursa is at times so great that the cloudy fluid from the bursa may push back the plunger of the syringe* The second needle is injected at site number two (just posterior to the

greater tuberosity when bone can be felt at the tip of the needle ... needle is slowly withdrawn for approximately ⅛ inch, and its tip pointed

in the direction of the assumed position of the first needle, the bursa is entered when the second needle has not been inserted for more than ½ inch Approximately 2 cc. of procaine is used in the insertion of each needle.

Both needles are now within the bursal sac. The 20 cc. syringe is connected to either of the needles and sterile saline is injected into the bursa; this is forced out of the other needle, washing out with it the calcium salts from within the bursa. The amount of saline required to wash the bursa clean may vary from 30 to 60 cc *The acute bursal pain often disappears following the washing out with even the first syringe full of salt solution*

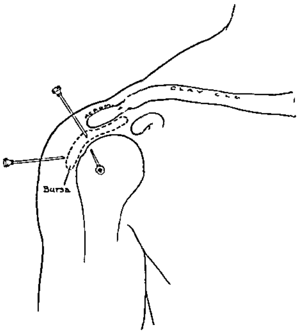


FIG 301 —Technic of procaine injection of the subacromial bursa and adjacent capsule of the shoulder joint (Haggart and Allen, courtesy of Surg Clin North America.)

When the bursa has been washed clean, the needles are removed and the area is covered with a sterile dressing The patient may now move his arm freely in all directions. The period of disability is shortened if care is taken to avoid the injection of the salt solution into the tissues around the bursa *This therapy is contraindicated in patients with old calcification and a hard bony-like appearance of the calcified area*

Multiple Punctures of the Bursa.—Flint first suggested needling the subdeltoid bursa in 1913 in cases of acute traumatic subdeltoid bursitis [29] More than thirty years ago Weeks inserted a hypodermic needle over the swollen region of the subdeltoid bursa (in a patient with a painful shoulder) and removed several cc. of clear straw colored fluid with almost immediate relief of pain [86] No additional treatment was given and the patient was never again troubled with pain in the shoulder Because of

this experience, he and his colleagues attempted to aspirate the bursa in these cases and found that the mere passage of the needle through the bursa relieved the symptoms whether fluid could be withdrawn or not *Simple multiple punctures of the bursal sac resulted in relief of symptoms in a large percentage of their patients.*[85] In their experience, the presence or absence of calcareous material made little difference in treatment as the calcified area subsequently disappeared.

This procedure is simple and can be performed without difficulty by anyone who will observe surgical asepsis and will acquaint himself with the anatomy of this region. All that is done is to infiltrate the skin and subcutaneous tissue over the bursa (usually the point of maximum tenderness) with 1 or 2 per cent procaine (using from 5 to 15 cc.). An intramuscular needle (gauge 17) is introduced into this area through the line of the procaine injection and is passed into the bursa, if fluid is encountered, it is evacuated (from a few minims up to several cc may be obtained), whether fluid is obtained or not, the bursa may be needled by a fan-wise series of punctures, and a sterile dressing applied. The patient is given an analgesic such as aspirin 0.6 gm (10 grains) and codeine 15 to 30 mg. ($\frac{1}{4}$ to $\frac{1}{2}$ grain). Pain may disappear within fifteen minutes after such treatment; however, one should order mild sedation for the ensuing twenty-four hours.

Simple Procaine Injection.—Procaine injection of the bursa and supraspinatus tendon has been used widely in recent years in the treatment of subdeltoid bursitis.[83] The results obtained in this as well as the previous procedure may be due to an increased local circulation, which aids in the more rapid removal of the calcified deposit, in addition, by puncturing the supraspinatus tendon, some of the calcified material may rupture into the bursa. Rupture of the calcified material from the [illegible]

Because of the intense pain which is experienced in the shoulder region, some of these patients will not permit any operative procedure on the shoulder without anesthesia. Some workers have used intravenous sodium pentothal or cyclopropane gas anesthesia, while others have found brachial plexus bloc to be an excellent anesthesia. These workers inject at least 20 cc of a sterile 1 or 2 per cent procaine solution into the bursa, and in addition, attempt to inject the capsule of the shoulder joint and the tendon in the region of the calcified deposits Such injections have proved very helpful in the acute cases, as well as in many of the more chronic adhesive types.

Local Physical Therapy.—Methods of local physical therapy which have been recommended in the treatment of acute subdeltoid bursitis include: (1) *local heat*; (2) *cold applications*; (3) *diathermy*; (4) *galvanization*; (5) *iontophoresis* (with mecholyl or magnesium sulfate)

Reports concerning the use of local forms of physical therapy vary markedly in both the methods employed and the results obtained[52,74] Some recommend dry heat and diathermy. In the experience of others, these result in increased pain during the acute stages of a bursitis; local applications of moist heat, rest in bed, and morphine 15 mg ($\frac{1}{4}$ grain) as necessary for pain, have produced better results for some workers (during the acute stages).

Coulter advocates daily treatments (twenty minutes each) for several weeks with **short wave diathermy** for acute subacromial bursitis, he also advises **infra-red radiation** for thirty minutes at least twice a day with the arm in abduction.[16] In some instances diathermy seems to aggravate the pain; it should be discontinued in such cases, and rest in bed with continuous moist heat to the shoulder may be tried. From ten to twenty or more of these treatments may be necessary for relief.[82]

Solomon and Morton found that *roentgen therapy and diathermy were*

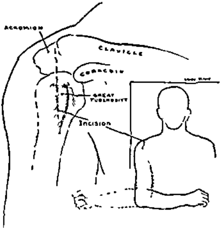


FIG 302 —Incision for exploration of the subacromial bursa Rotation of the forearm (see insert) permits visualization of the short rotator muscles (Haggart and Allen, courtesy of Surg Clin North America)

then repeated until the patient had received about fifteen minutes of treatment In subsequent treatments, the total treatment was extended to a maximum of twenty minutes In the more chronic cases, the current flowed continuously for twelve to fourteen minutes for the first treatment and subsequent treatments were increased to a maximum of twenty minutes Treatments were given three times a week

Echtman and others advocate the use of *cold wet compresses or an ice*

(See also chapter on Physical Therapy)

Exploration and Drainage in the Treatment of Subdeltoid Bursitis.— Some surgeons believe that the most rapid relief in the treatment of

subdeltoid bursitis follows exploration of the bursa and removal of the calcified material.[5] The operation is simple, being performed under anesthesia (local infiltration, brachial plexus block, or general anesthesia if desired). By means of a small vertical incision over the region of the anterior surface of the greater tuberosity (1 inch or more in length), the fibers of the deltoid are separated and the roof of the bursa may be pricked with the point of a knife and opened. The calcified substance can then be seen through the floor of the bursa, if this is pricked the toothpaste-like material is allowed to exude This may be curetted gently if desired. (*Thorough curettage is unnecessary*) The muscle wound is gently sutured with catgut and the skin is closed.



Fig 303—*Simple traction apparatus for increasing shoulder motion* (Swaim and Kuhns, courtesy of Jour Am Med Assn)

The postoperative care of the patients includes either rest in bed for a period of one or two days, or carrying the affected arm in a sling. In either case, *one of the most important points in follow-up treatment is the use* [illegible]

Special exercises to develop shoulder muscle power [illegible] advantage by Haggart and Allen These include swinging the arms anteroposteriorly and also mediolaterally with the patient in a stooped position; placing the hand of the affected arm on the opposite shoulder posteriorly; touching the floor without bending the knees, and holding the arm out horizontally so that the fingers just touch the wall and then attempting to [illegible]

[illegible] *of after treatment should* [illegible]

Regardless of the method of treatment of the [illegible]

follow this with exercises, local heat and massage; the physician may be amazed by the rapid return of mobility and power in the shoulder joint, and by the absence of local muscular atrophy in this region.

Cortisone, ACTH and Hydrocortisone.—Hormone therapy, using cortisone or ACTH, though not always beneficial, has been of distinct value in the treatment of acute or subacute bursitis with or without calcium deposits. Conclusions at present must be guarded as no adequate series of patients has been reported and the natural tendency for acute bursitis to clear spontaneously makes the assessment of any therapy difficult. However, reports would indicate that 50 to 60 per cent of acute or subacute cases are relieved of pain in about two days with subsequent increase in range of

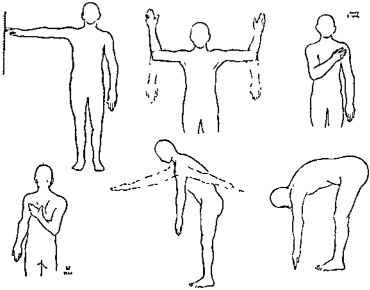


FIG 304.—Exercises useful in redeveloping shoulder muscle power, of special value are climbing the wall with the fingers of the outstretched abducted arm, and swinging the arms anteroposteriorly and also mediolaterally (not shown) with the trunk flexed (Haggart and Allen, courtesy of Surg Clin North America)

movement, using a schedule of dosage similar to that for rheumatoid arthritis. In chronic bursitis, (frozen shoulder) the results are less dramatic and maintenance therapy may have to be continued for several months, otherwise the pain and disability tend to recur.[79] Prompt relief following the injection of 15 to 25 mgms of hydrocortisone in acute bursitis has been reported by Hollander.[40] Results in chronic bursitis were much less satisfactory. It is obvious that steroid therapy will have a place in the treatment of all phases of bursitis, but its exact position on the list of conservative measures, must await further study.

Treatment of Chronic Bursitis.—Patients with chronic subdeltoid bursitis without calcification are *not* definitely benefited by surgical intervention, (chronic cases with calcified deposits are usually relieved by operation). These patients usually complain of chronic shoulder pain without much

limitation of motion. The pain is frequently more noticeable at night and prevents the patient from lying on the affected side. It may also be present during movements requiring abduction of the arm, such as putting on a coat. Ferguson has observed in some of these patients a radiation of pain down the arm and into the fingers with some associated swelling and stiffness.

Physical signs in these patients include:

1. *Tenderness* on deep pressure over the greater tuberosity; this is usually diagnostic.
2. *Pain on elevation of the arm* or in lowering the arm from a position of elevation. This occurs simultaneously with the passage of the greater tuberosity under the edge of the acromion process.
3. Small *calcium deposits* (more common in younger than in older patients).
4. Occasionally, *decalcification of the greater tuberosity* of the humerus with bony excrescences and cortical eburnation (in older patients).
5. A click or crepitation on elevating or lowering the arm; the click is simultaneous with the occurrence of pain. This is present in some of the older patients only and is more characteristic of rupture of the supraspinatus tendon.

Good results have been obtained in the treatment of chronic bursitis by means of roentgen-ray therapy in about 50 per cent of patients.[13,67] Pendergrass, Hodes and others advise one or two treatments a week, extending over a period of from four to six weeks. Symptoms may be exaggerated *for a period of twenty-four hours following roentgen therapy. If patients with chronic bursitis with calcification* do not respond to roentgen therapy or diathermy, one may advise *incision and surgical curettage of the calcified area. If this is done properly, practically all of these patients will be relieved.*

If roentgen therapy does not produce relief, and if the patient refuses surgery, one may prescribe diathermy and rest of the shoulder.[36,70] Rest of the shoulder is best carried out in these cases in a position of abduction.

Many physicians have seen no marked beneficial response of shoulder symptoms following removal of focal infection; occasionally, the later has immediately preceded the stage of comfort. If definite focal infection [illegible] general health measure, but the [illegible] removal of [illegible]

CHRONIC ADHESIVE BURSITIS

In this group are placed those patients in whom adhesions have formed between the bursal surfaces so that the gliding mechanism of the bursa has been lost. This form of bursitis shows the poorest response to treat- [illegible] therapy in these cases is to separate the adhesions [illegible] 'frozen shoulder," [illegible] tendinitis, etc.

In chronic adhesive subacromial bursitis, there is proliferation of fibrous tissue in the periarticular structures, hypertrophied synovial folds and dense adhesions within the adjacent bursæ.[8] The surrounding muscles

the subacromial bursa which result in loss of the normal gliding function are probably the basic factor causing disability in chronic adhesive subacromial bursitis.

Symptoms of Chronic Adhesive Subacromial Bursitis
Marked *atrophy* of *the deltoid and spinatus muscles*, often associated with coldness and stiffness of the hand and atrophy of the skin
Inability to move the arm through its normal motions at the shoulder (particularly abduction and external rotation)
An aching *pain in the shoulder*, sometimes radiating to the insertion of the deltoid or to the hand
Tenderness over the greater tuberosity of the humerus and usually along the deltoid muscle as far as its insertion
Roentgen evidence of atrophy of the greater tuberosity and sometimes the presence of a calcified area in those cases which have followed an acute bursitis

The *symptoms* of chronic adhesive subacromial bursitis include *persistent pain* in the shoulder which may extend into the neck or down the arm to the hand. There is *marked loss of shoulder joint function*. In some instances, the only complaint is the latter. If the process has been present for some time, there is some degree of *atrophy of the shoulder muscles* and in some instances one notes *loss of finger joint function* with swelling of the hand and fingers. *Lateral abduction and external rotation are the particular shoulder motions that are lost.* There is usually an almost normal range of anteroposterior motion as well as internal rotation. *The points of tenderness over the shoulder are less acute and less poorly localized than in patients with an acute bursitis.* Tenderness is defined more accurately in the chronic adhesive

forbids function of the shoulder joints, such as prolonged rest in bed following hemiplegia,[22] cataract operation, coronary occlusion, etc.

If the symptoms are mild and there is only moderate restriction of

include abduction, internal and external rotation.

tinued; the results are usually fairly good. Several manipulations are usually necessary in advanced cases. It is better to do too little, rather

61

Important Pointers in the Subdeltoid Bursitis "Syndrome"

Synonyms: Subacromial bursitis, periarthritis of the shoulder, painful shoulder, supraspinatus tendon calcifications, etc. By common usage, the term "subdeltoid bursitis" includes in addition to bursitis the frequently accompanying supraspinatus tendon calcifications.

Usual Pathology Inflammation in the general region of the subdeltoid bursa frequently with calcification (not in the bursa as a rule, but in the tendons of the supraspinatus or of the other short rotators).

Etiology. Most commonly from acute or chronic trauma to tendons of the short rotators

Outstanding Symptoms Severe pain in the shoulder, tenderness over the greater tuberosity of the humerus, inability to elevate the arm because of severe pain, and often the presence of calcified material seen by roentgen-ray

Types: Acute traumatic, acute non-traumatic (probably from calcium deposits), chronic (with and without calcium deposits).

Treatment In general, best results are obtained *in the acute and subacute*

[illegible]

tion (usually under pentothal sodium anesthesia). Exercise for the shoulder joint must be instituted as soon as possible in all cases after cessation of the acute pain. Surgical curettage of a calcified area may be necessary occasionally.

Results of Therapy Improvement occurs in most patients, often even without treatment, in cases without treatment, symptoms usually persist for long periods. Relief of acute pain (usually within twenty-

[illegible]

than too much, at one manipulation This procedure produces an increase in pain in the shoulder for a period of twenty-four hours following the manipulation After breaking up these adhesions, exercises for the shoulder (as described previously) form an important follow-up treatment Recovery practically always occurs, but is usually measured in months, rather than days

Codman did not favor rupture of these adhesions under anesthesia, or open operation for the release of adhesions He obtained *best results* in this condition *if the patient was put to bed and gradual external rotation and abduction of the arm was produced* by applying a splint to the forearm, each end being tied to the head of the bed. He also used morphine, as necessary, and found that within a day the adhesions usually yielded, the tuberosity moved under the acromion and the arm became comfortable The patient was kept in bed for one to two weeks with the arm abducted until the shoulder motions were free in all directions. During this period

he was allowed out of bed each day for swinging exercises (as previously described) while in the stooped position.

Summary of Subacromial Bursitis

- Most cases of subacromial bursitis recover spontaneously but the duration varies from a few weeks to two or three years. In many of the attacks it is so severe that they seek immediate treatment[11]
- Caldwell and Unkauf reviewed 340 cases and found that calcium deposits occurred in 39 to 47 per cent with no difference in incidence in the acute, subacute or chronic state. They found that most patients become reasonably comfortable and gain a fair use of their arm within a few weeks with conservative, non-operative measures
- The principal aim is to relieve the acute pain and then preserve motion or overcome adhesions by heat and stretching exercises
- In acute cases, they found that Procaine injection, x-ray therapy or [illegible]
- In [illegible]
- In chronic cases, no good results followed manipulation but Procaine injections, x-ray therapy, and physiotherapy appeared to give equal benefit
- In all stages, the combination of x-ray therapy and physiotherapy gave the best results
- Conservative measures were satisfactory in 70 to 85 per cent
- X-ray therapy relieved the pain in 85 per cent
- Physiotherapy is a necessary adjunct with all forms of treatment and is satisfactory alone in subacute and mild cases
- The longer the duration of symptoms, the poorer are the results from conservative therapy[11]
- The exact status of hormone therapy has not yet been determined but preliminary reports suggest that cortisone, ACTH, and local hydrocortisone are useful in the treatment of all stages of the disease but are less satisfactory in the chronic case
- Surgical treatment should be restricted to cases with large calcium deposits, and those having recurrent attacks which fail to respond to conservative measures

RUPTURE OF THE SUPRASPINATUS TENDON

Rupture of the supraspinatus tendon is a frequent cause of disability of the shoulder. Codman believes that complete rupture of the supraspinatus [illegible] accidents [illegible] of minor [illegible] complete [illegible] verse tear through all of the fibers of the supraspinatus tendon, in addition to the floor of the bursa lying over it, and the roof of the joint capsule on its under surface. This condition is very infrequent in women.

Rupture of the supraspinatus tendon may be produced by muscular strain when the shoulder is raised in abduction or by a direct blow on the shoulder, as from a fall.

Its *symptoms and signs* include:

1 History of *injury* (usually a fall).

[illegible] in

4 *Tenderness over the greater tuberosity* of the humerus.

5 Negative roentgen-ray findings.

The sudden pain at the onset of the injury is probably due to the tear of the supraspinatus tendon close to its insertion on the greater tuberosity. Recurrence of pain after a free interval is *thought to be caused by distention* of the tissues (joint, bursa, etc) in this region by blood. In some patients, the pain may not be severe and only loss of power of abduction of the shoulder will call the patient's attention to his *illness, however, pain* is the predominating symptom in most of these cases Codman has called



FIG 305 —Double cervical ribs at *A* (Holmes and Robbins)

attention to the presence of a sulcus, an eminence and tenderness at the *insertion of the supraspinatus, with a velvety, soft crepitus as the tuberosity* disappears under the acromion when the arm is elevated The sulcus is usually large enough to admit the tip of the finger.

Treatment.—*In every case of complete rupture of the supraspinatus tendon, the treatment is operative,* consisting of suture of the divided tendon [19,30] Excellent results have been reported [88] Postoperative treatment includes *an axillary pad with the arm in slight abduction; muscular exercise* and massage may be begun a few days postoperatively.[87]

Partial ruptures of the supraspinatus tendon may heal without operative *intervention* The most common form of *treatment for partial rupture* is fixation of the arm in abduction for five to six weeks by splints or plaster dressing to relax the tendon and approximate the torn ends, *in addition* to massage and stooping exercises The patient should not use the arm when standing until he can swing his arms freely (and without pain) in the stooping position. *Lateral or anteroposterior pendulum-like* motions

can be performed without much muscular effort when the patient is in the stooping position; in addition, the weight of the arm assists in stretching any contracted tissue about the joint

CERVICAL RIB AND SCALENUS ANTICUS SYNDROME

Symptoms caused by a cervical rib or pressure of the scalenus anticus muscle on the brachial plexus have led to confusion in diagnosis of patients with shoulder pain.[60]

Cervical Rib.—This is a fibrous, cartilaginous or bony rib extending outward from any of the cervical vertebra from the second to the seventh *Cervical ribs* are present in about 1 per cent of all individuals[21] From 10 to 45 per cent of patients with cervical ribs exhibit symptoms from these.[2]

Palpation of cervical ribs is not an easy procedure in many cases, roentgen examination should always be made if the symptoms of cervical rib syndrome are apparent. Even after the roentgen-ray has demonstrated a cervical rib, palpation is at times difficult.

The Scalenus Anticus Muscle *runs from the transverse processes of* the third, fourth, fifth and sixth cervical vertebræ downward and outward (at an angle of about 30 degrees with the spine) over the cords of the brachial plexus and the subclavian artery to its insertion on the scalene tubercle of the first rib

Symptoms Produced by Cervical Ribs or by Spasm of the Scalenus Anticus Muscle.—The cords of the brachial plexus may be irritated by

Symptoms of Cervical Rib and Scalenus Anticus Syndrome
Neuralgic pain extending from the neck to the hand (usually on the ulnar side)
Tiredness or weakness of the extremity
Heavy dragging sensation in one or both shoulders or pain over the deltoid extending down the arm and often centering about the elbow.
Tenderness over the lower third of the scalenus anticus muscle immediately above the clavicle
Paresthesis or anesthesia in a similar area, occasionally areas of hyperesthesia
Symptoms usually made worse by work and exercise
[illegible]
Presence of a bruit over the clavicle
[illegible]

being stretched over the sharp edge of the cervical rib, resulting in a friction neuritis;[45] in the presence of a cervical rib, the scalenus anticus muscle may compress the subclavian artery or brachial plexus against this rib. The lower nerves of the brachial plexus are more commonly involved.[66] As the scalenus anticus muscle is innervated by the brachial plexus, irritation of the brachial plexus may result in a spasm of this muscle, which in turn produces further compression of the plexus, forming a vicious circle. In patients in whom no cervical rib is present, the scalenus anticus muscle may elevate the first rib to an abnormal extent and thus interfere with the brachial plexus and subclavian artery; the symptoms resulting from such an action of the scalenus anticus muscle are identical with those of a cervical rib. Symptoms may also be the result of an abnormally low position of the shoulder,[17] high fixation of the sternum and ribs, low origin of the brachial plexus, lesions of the cervical spine and traumatic or infectious lesions of the shoulder girdle.[32]

Age at Which the Symptoms of Cervical Rib Usually Appear.—The average age of appearance of symptoms is about thirty years.[69] When the shoulder girdle is high (as in childhood), symptoms are less prone to [illegible]. There is a progressive descent of the shoulder girdle which is not [illegible] posture may result

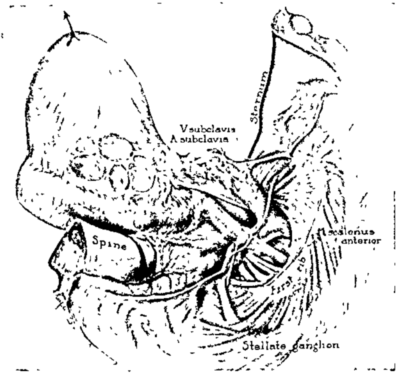
The pain usually begins in the neck and spreads down the arm as far as the hand; in some cases, the pain may be noticed first in the hand and then radiates upward into the neck and chest. In some patients, the pain radiates up the neck and over the scapula.

In about 4 per cent of the patients with cervical ribs, a dilatation of the subclavian artery occurs just beyond the point of constriction. *Pressure on the brachial plexus by cervical ribs or the scalenus anticus muscle may cause pain simulating angina pectoris or coronary artery occlusion.* The pain of brachial plexus pressure may be experienced in the anterior aspect of the chest or precordium. This pain may also be induced by exertion (as in some cases of angina pectoris), however, only certain forms of exertion (such as pulling the shoulder downward or turning the head to one side) will precipitate the brachial plexus pain. The continued carrying [illegible]

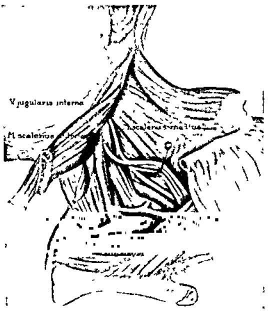
[illegible]

the anterior scalenus muscle.[2] In 1938, Naffziger pointed out [illegible] tightened scalenus anticus muscle could cause compression of the subclavian artery and that the latter could cause pain in the shoulder and hand. Freiberg suggested (1938) that spasm of the scalenus anticus muscle might be a common complication of many common lesions about the [illegible] shoulder [illegible]

of symptoms



A



B

FIGS 306 *A* and *B* —Dissection of the neck to show the arrangement of the brachial plexus, subclavian artery and the scalenus anticus muscle (Naffziger and Grant, courtesy of Surg , Gynec and Obst)

The *etiology of the scalenus anticus syndrome* is compression of the brachial plexus which develops in some individuals because of abnormal development of the shoulder girdle or of the brachial plexus permitting friction or pressure on the brachial plexus and subclavian artery. This pressure or friction irritates some of the fibers supplying the scalenus anticus and scalenus medius muscles, causing abnormal elevation of the first rib

Compression of the brachial plexus as in the scalenus anticus syndrome results in *pain, numbness and tingling which may extend from the cervical, scapular or pectoral regions to the hand and fingers. The pain is usually dull, gnawing or burning* and is affected by work and posture. Coldness and cyanosis of the fingers are infrequent.

The medial cord of the brachial plexus is usually affected, resulting in pain primarily in the arm between the shoulder joint and elbow following the sensory distribution of the ulnar nerve.[76] *Scalenus tenderness* is a constant finding. *Symptoms may vary from an excruciating pain to a mild, burning sensation in the shoulder, arm and finger.*[46] Most of these patients complain that their symptoms are *worse* or are severe *at night* while in bed.

Pain in the shoulder may produce spasm and contractures of the muscles of the shoulder girdle; the scalenus anticus muscle often takes part in this spasm. Spasm or marked hypertrophy of the scalenus anticus may cause direct pressure on the brachial plexus or indirect pressure by unduly elevating the first rib (which latter then presses into the brachial plexus and subclavian artery

When the shoulder

normal, leading to sym

Conservative Therapeutic Measures.—Keep the arm above the shoulder with the hand behind the head. Such elevation of the arm and shoulder girdle displaces the brachial plexus upward out of the scalenus triangle and relieves the tension upon the nervous and vascular structures. *Heat* should be applied over the region of the scalenus muscle

Theis reported that 17 of 20 patients were improved merely by cor-

laced with one long edge at an angle of 45 degrees to the top of the bed, the second was placed at right angles to the first with one-half of this pillow overlapping the top half of the first. A third pillow was placed horizontally (parallel to the top of the bed) directly on top of the upper ends of both number one and

number two pillows The patient's head then rested upon pillow number

shoulder rested upon the mattress between the pillows. These pillows were so arranged as to force the shoulders, head and neck forward when the patient was on his back, and to prevent sinking of the head toward the under shoulder when the patient was on his side

Many patients with scalenus anticus syndrome have learned that sleeping with the arms over the head relaxes the squeezing mechanism which compresses the brachial plexus between the scalenus anticus and medius muscles and the first rib; although this position usually affords relief, it is rarely maintained while the patient is asleep.

Gage produced *temporary relief of scalenus spasm by procaine injection into the muscle* and suggested that this might be of importance in identifying *the syndrome.*[23]

Relief of pain after procaine injection of the scalenus anticus usually appears in two to five minutes, the maximum effect being observed in ten to twenty minutes.[47] If one happens to also inject the cervical sympathetic fibers, redness of the eye and lacrimation may be noticed on the side injected, together with enophthalmos, drooping of the eyelid and constriction of the pupil; this usually disappears within forty-five to ninety minutes.

performed

lar impairment; early surgical intervention will result in anatomical and functional recovery.[110]

Technic of Scalenus Anticus Injection[46]

1. Turn the patient's head and chin to the non-affected side
2. Place the tip of the left middle finger against the outer border of the

straddle the first rib near the insertion of the scalenus muscle It may be necessary to alternately flex and extend the head with the free hand to aid in following the course of the scalenus muscles

5.

6.

7.

8. The plunger of the syringe must be withdrawn before injecting to be sure that the point of the needle is not in a blood-vessel.
9. The needle is usually pointed almost directly posterior, toward the transverse processes; if contact with the processes is made, the needle is withdrawn slightly before injection is begun

Treatment of Cervical Ribs.—Cervical ribs may vary in size from merely
one of the lower cervical vertebræ

available include: Conservative orthopedic measures.

Section of the scalenus anticus muscle.

Removal of the cervical rib.

In many of the patients, symptoms are mild. These individuals may not submit to operative procedures. These patients are often subject to remissions and exacerbations. If the symptoms are not severe, attempts should be made to elevate the shoulder upward and backward by means of a sling, and to develop the trapezius and levator anguli muscles by means of physical therapy *Corrective exercises and good posture* may be helpful in some patients. Repeated injections of procaine may give prolonged or even lasting relief, these should not be performed too often as they may lead to scar tissue formation or possibly damage to the sympathetic nerves from too deep an injection

If more radical treatment is required, the choice is section of the scalenus anticus muscle; this has produced favorable results.[29] Tenotomy of the scalenus anticus muscle has replaced the older operation of resection of the cervical ribs in many instances.[1,33,50] The operation of tenotomy is much safer, gives better results than rib resection and is not accompanied by unfavorable postoperative sequelæ. In some cases, however, in which there is evident pressure from the cervical rib or its tendinous attachment, resection of this rib and its attachment may be necessary. When large

cardiac disease, congenital deformities, apical pulmonary tumors, etc).

SHOULDER LESIONS IN BASEBALL PITCHERS

Bennett described the lesions which may occur in the shoulder and elbow in professional baseball pitchers. He emphasized that the pitcher is subject to all the disabilities of the shoulder to which the average person is, such as *subacromial and subdeltoid bursitis, irritation of* the supraspinatus, irritation of the biceps tendon, traumatic synovitis and the inflammatory diseases [7]

In the pitcher, the changes are most often seen in the *anterior group of disorders, i. e*, inflammatory and traumatic lesions of the supraspinatus tendon, the *subacrominal* bursa, the subdeltoid bursa, the biceps tendon and the coracoid bursa. With rotation of the head of the humerus, the supraspinatus tendon may be rubbed against the acromion producing traumatic irritation or fraying of the tendon The biceps tendon may be injured by the same motion Trauma between the head of the humerus

traumatized at the

humerus is pressed against the coracoid process The supraspinatus is irritated by the overhand pitcher while the coracoid bursa is irritated by the sidearm pitcher.

The anterior group of lesions produces sensitiveness about the point of insertion of the supraspinatus and discomfort on abduction of the arm If there is involvement of the biceps tendon, one notes sensitiveness in the region of the bicipital groove and discomfort when the forearm is flexed against resistance. Crepitus may be elicited on active motion if these tendons are frayed by trauma.

In the simple traumatic group, rest and local heat form the basis of treatment; most of these traumatic injuries respond to conservative care If the tendons become frayed, the biceps may respond to surgical treatment but one could not expect a reconstructed supraspinatus tendon to perform the accurate and strenuous function required in a professional pitcher

Lesions in the posterior part of the shoulder shorten the career of the professional pitcher. Due to excessive use of the arm and marked pull on the posterior capsule and triceps tendon, *a deposit of bone may develop on the posterior inferior border of the glenoid fossa.* This deposit may irritate the capsule and synovial membrane as well as the circumflex nerve producing referred pain to the deltoid region Symptoms of these lesions include local discomfort and sensitiveness in the posterior portion of the shoulder, in addition to pain referred to the deltoid[7] With such a lesion, the pitcher cannot throw hard after an inning or two and must leave the game This lesion does not cause discomfort during normal motion of the shoulder or even on throwing at moderate speed Although it is possible to remove this exostosis, it is questionable whether this should be done as it does not cause symptoms except on hard throwing, and it is doubtful whether the professional pitcher can resume his occupation after such an operation.

LESS COMMON CAUSES OF PAINFUL SHOULDERS

[illegible]

aortitis, aneurysm, coronary occlusion, pneumonia, pleurisy, pneumothorax, pulmonary neoplasm, gall bladder disease, cervical Pott's disease, herpes zoster, etc), tumors of the spinal cord and nerves, peripheral neuritis, discogenetic disease, etc. Shoulder pain arising from *arthritis of the cervical spine* is not uncommon and has been discussed in the section dealing with arthritis of the spine *Calcified lymph nodes* in the neck may produce shoulder pain by irritation of the brachial plexus (mechanically or by actual inflammatory spread)

SHOULDER-HAND SYNDROME

One commonly sees persistent pain in the shoulder region as an aftermath of myocardial infarction or in patients with severe and frequent attacks of angina pectoris[4,9,24,25,41,55,57]

In some cases the hand is also affected Askey[4] reviewed the syndrome of painful disability of the shoulder and hand following coronary occlusion

and described 22 cases, 11 men and 11 women from forty-eight to seventy-nine years of age.

One week to seven months after coronary occlusion or persistent angina the patients developed acute or subacute periarthritis of one or both

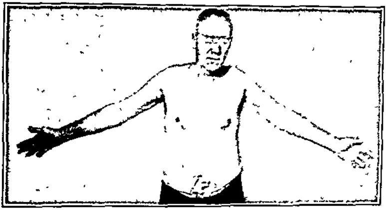


FIG 307 —Shoulder-Hand Syndrome: Inability to abduct at shoulder joints, contracture of fingers



FIG 308.—Shoulder-Hand Syndrome X-ray showing the marked osteo-porosis in bones of hands and wrists

one or
ffected
s Con-
tracture developed in seven of ten cases examined for it. A consistent

point of tenderness was found over the trapezius branches of the cervical plexus The shoulder disability lasted an average of six months but residual stiffness usually resulted. Various treatments given by Askey were valueless, including salicylates and paravertebral injections of alcohol into the first and second dorsal sympathetic ganglia

Six to eight weeks after myocardial infarction hands and wrists were affected by "periarthritis" in 3 cases of Meyer and Binswanger [57]

The shoulders or hands or both were affected in 39 of 178 consecutive cases of myocardial infarction studied by Johnson[41] who termed the condition "post-infarction sclerodactylia " In these 39 cases the onset occurred three to sixteen weeks after the infarction and all the patients were referred

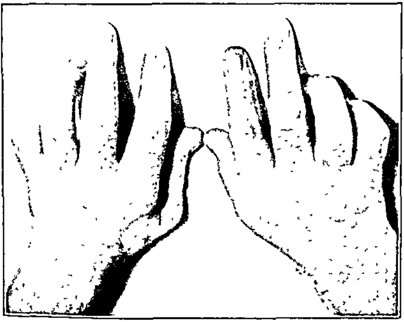


FIG 309 —Shoulder-Hand Syndrome Dorsal view of hands showing trophic changes in skin, flexion contractures of fingers

as cases of arthritis. Pain, limited motion, atrophy, and palmar contrac-

occlusion

Steinbrocker[77] reported 6 of 200 patients with the "shoulder-hand syndrome" with no apparent precipitating cause He stressed the fact that these are usually diagnosed as atypical rheumatoid arthritis Other

separate entity [78]

Steinbrocker described 3 phases. (1) Painful disability of the shoulder with generalized swelling and stiffness of the fingers, (not confined to joints) appearing acutely or insidiously over three to six months The shoulder or hand may be affected first or both simultaneously. (2) Over a period of three to six months there is gradual relief in the shoulder and usually a decrease in swelling in the hand, but stiffness and flexion deformity of the fingers becomes more pronounced. Osteoporosis of the hand becomes increasingly distinguishable. (3) Trophic changes occur in the hand with palmar fascial contractures which may last for years.

The mildest case was better in ten months, the worst case still had shoulder and hand disability after seven years The sedimentation rate was elevated in one case only.

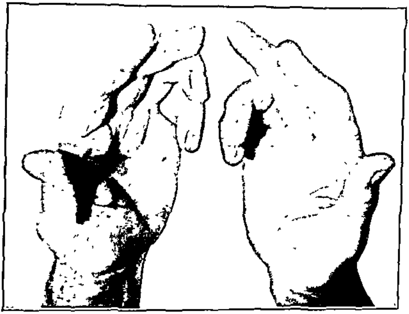


FIG. 310 —Shoulder-Hand Syndrome Bilateral palmar fascial contractures resembling Dupuytren's Contracture.

Similarly Oppenheimer[64] described 14 patients with pain, swelling and atrophy in one hand and a history of pain in the shoulder girdle of the affected side. In all patients *x*-rays showed narrowing of one or two intervertebral foramina of the upper cervical spine confined to the diseased side. There were no arthritic manifestations in the hand. Oppenheimer considers the syndrome a trophic disorder caused by pressure upon the cervical nerve roots within the intervertebral foramina.

The shoulder hand syndrome also may occur after hemiplegia, splinting of an arm after fracture, manipulation of a shoulder or in many cases spontaneously without apparent cause.[37]

Steinbrocker[77] feels that the syndrome may represent variations of sympathetic and spinal reflex reactions to different etiological factors, some of which are known, trauma, myocardial infarction, cervical osteoarthritis, etc

No treatment has been completely successful but hormone therapy would appear to be more beneficial than any previous measures. Steinbrocker,[18] treated 10 patients with cortisone or ACTH who previously had failed to respond to physiotherapy, manipulative therapy or local and stellate ganglian blocks. One patient had complete recovery, 5 great [illegible]

75 to 100 mgms. of ACTH. Maintenance therapy may be required for several months and contractures already existing are not influenced by the drug.

Along with hormone therapy, heat and exercises should be carried out to maintain movement and severe cases may require splints to prevent deformity. Wax baths are helpful for the hands and elevation of the limb will reduce edema The use of paravertebral sympathetic nerve blocks has been largely supplanted by hormone therapy and the use of tetraethylammonium chloride, which produces a blockade of the autonomic ganglia is no longer worthy of general use

BIBLIOGRAPHY

1 [illegible]

5 Bartels, W [illegible]
6 Bates, W [illegible]
7. Bennett, G [illegible]
8 Bennett, R [illegible]
9 Boas, E. P, and Levy, H Am Heart Jour, 14, 540, 1937
10 Bosworth, [illegible]
11. Caldwell, [illegible]
12 Carnett, J. [illegible]
13 Canigiani, [illegible]
14 Codman, E A The Shoulder, Boston, Thomas Todd Company, 1934
15 Cooper, [illegible]
16 [illegible]
17 [illegible]
18 [illegible] it Surg, 19, 993, 1937
19 Davis, T W, and Sullivan, J E Ann Surg, 106, 1059, 1937
20 Dick, G F, Hunt, L W, and Ferry, J L Jour Am Med Assn, 116, 1202, 1941
21 Donald, [illegible] rg, 111, 709, 1940

[illegible] r Med Sci, 191, 201, 1936
25 Epstein, A C, and Kinnell, J Arch Int Med, 66, 890, 1940
26 Ferguson, L K, Internat Abstr Surg, 66, 472, 1938
27. Fisk, G H Can Med Assn. Jour, 1, 213, 1944
28 Flint, J M Jour Am Med Assn, 61, 1224, 1913
29 Flothow, P. G West Jour Surg, 44, 570, 1936
30 Fowler, E B Jour Am Med Assn, 101, 2106, 1933
31 Freiberg, J A Jour Bone and Joint Surg, 4, 860, 1938
32 ———. Jour Bone and Joint Surg, 20, 860, 1938
33 Gage, M Surgery, 5, 599, 1939
34 Graham, E A, Singer, J J, and Ballon, H C Surgical Diseases of the Chest, Philadelphia, Lea & Febiger, p 87, 1935
35 Haggart, G E, and Allen, H A Surg. Clin North America, 15, 1537, 1935

36. Harris, J. F.: Jour. Am. Med. Assn, 81, 98, 1923.
37. Hench, P. S., *et al* Ann. Int Med, 28, 66, 1948
38 Henschen, C, and Heusser, H.: Chirurg, 9, 266, 1937.
39. Hitzrot, J M: Ann. Surg, 98, 273, 1933.
40 Hollander, J. L, Brown, E. M, Jessar, R. A, and Brown, C. Y.. Jour Am Med Assn, 147, 1629, 1951.
41. Howard, T.. Med. Jour. and Rec., 131, 364, 1930
42. Howarth, M B: Surg Gynec. and Obst., 80, 337, 1945
43 Inman, V. T, Saunders, J. B de C. M, and Abbott, L C: Jour. Bone and Joint Surg, 26, 1, 1944.
44 Johnson, A C: Ann. Int Med, 19, 433, 1943.
45 Jones, R, and Lovett, R. W.: Orthopedic Surgery, 2d ed, Baltimore, William Wood & Co, p 585, 1929
46 Judovich, B D, and Bates, W: Jour. Internat. Col Surg, p 26, Jan-Feb, 1942.
47 Kaplan, L.: Pennsylvania Med. Jour., 44, 289, 1940
48. ———: Pennsylvania Med Jour., 45, 37, 1941.
49. ———: Arch Surg, 42, 739, 1941.
50 Keen, W. W.. Am Jour Med Sci, 133, 173, 1907.
51 Kehl, K C: *Ann. Int Med*, 19, 213, 1943
52 Kovacs, R: Arch Phys Ther, 23, 341, 1942
53. Kuster, E. Verhandl d deutsch. Gesellsch. f. Chir., 31, 364, 1902.
54 Lattman, I.: Am. Jour Roentgenol and Rad Ther, 36, 55, 1936
55 Leech, C. B.. Rhode Island Med Jour., 21, 101, 1938
56 Meyer, A W.: Arch Surg, 35, 646, 1937.
57. Meyer, J C, and Binswanger, H. F.. Am Heart Jour, 23, 715, 1942.
58. [illegible]
59 [illegible]
60 [illegible]
61 [illegible]
62. [illegible] 38.
63 [illegible]
64 [illegible]
65 [illegible]
66 [illegible]
67 [illegible]
68 [illegible] 42
69 [illegible]
70 [illegible]
71. [illegible]
72. [illegible]
73. Silbert, S: Surgery, 7, 392, 1940
74 Solomon, W M. Brit Jour. Phys Med, 5, 16, 1942
75 Solomon, W. M, and Morton, J L Arch Phys Ther, 22, 607, 1941
76 Spurling, R G, and Grantham, E G.. Jour. Missouri State Med Assn, 38, 340, 1941.
77. Steinbrocker, O. Amer Jour Med, 3, 402, 1947.
78 Steinbrocker, O, Spitzer, N, and Friedman, H. H.: Ann Int Med, 33, 22, 1948.
79. Steinbrocker, O, *et al.* Bull. N Y. Acad. Med., 27, 560, 1951
80. Steindler, A: Northwest. Med, 40, 3, 1941
81. Theis, F V. Surgery, 6, 112, 1939
82 Titus, N E: Am Jour Surg, 6, 318, 1929
83 Travell, J, Rinzler, S, and Herman, M Jour. Am Med Assn., 120, 417, 1942
84 Voight, W W.: Illinois Med Jour, 75, 340, 1939
85 [illegible]
86 [illegible], 3, 40, 1936
87. [illegible] 1931.
88 [illegible]
89 ———: Brit Med Jour, 1, 1201, 1940
90. Wilson, S A. K: Proc Roy. Soc Med (Clin. Sect), 6, 133, 1912-1913

CHAPTER 55

DUPUYTREN'S CONTRACTURE

By Morris A Bowie, M D

Contraction of the palmar fascia was described by Plater in 1610, and Sir Astley Cooper described correction by fasciotomy in 1818 Dupuytren's classic description of fascial contracture, flexion deformities of the fingers, and loss of function appeared in the Lancet for 1834 [12] Reviews of the literature have periodically come forth [8,10,17,20,21,23,30,32,40,43]

Etiology.—The etiology of this condition is still unknown Numerous

i the most important are he-

nnective tissue originating in

disturbances of sympathetic

tone,[34] nervous system lesions, and various disease states Histological similarity to other fibroplasias such as keloids, fascial desmoids, and induratio penis plastica (Perrone's disease) have been noted [4,16,21]

Dupuytren's contracture has been observed in several generations in the same family, usually

been noted in several la

cases from fifteen author

per cent, in Skoog's own group of 50 cases involvement in other members of the family was shown to be present in 44 per cent In this series males were affected nine times as frequently as females [43] It might be assumed that hard manual labor might be related in view of this sex disproportion, but the majority of authors discount occupational trauma A curious, and perhaps significant, association between Dupuytren's contracture and epilepsy has been described by Lund [25] He reported contractures of the hand in 50 per cent of 190 male epileptics, and in 25 per cent of 171 females with the convulsive state Skoog subsequently studied 207 afflicted males and found palmar contractures in 42 per cent Concurrent hereditary predisposition toward both of these conditions may be the common denominator for this unusual association The most severe and resistant case seen by the writer was a middle-aged male epileptic in whom the palmar contracture was part of a reflex dystrophy involving the shoulder on one side, this patient's shoulder-hand syndrome was markedly improved when adequate anti-convulsive therapy was initiated

Increased sympathetic tone has been suggested as a cause,[33] and Hale

phenomenon A reflex dystrophy involving the shoulder and the hand has been observed with increasing frequency in recent years [2,23,30,46] This has been aptly termed the *Shoulder-hand syndrome* by Steinbrocker It is most frequently seen after myocardial infarctions, hemiplegias, splinting, minor injuries to the shoulder, manipulation, cervical arthritis, and occasionally

completely alone. Pain in the shoulder with associated swelling of the finger pads and palm may appear within a few weeks to a year after coronary occlusion. Gradually the shoulder disability improves, but the hand changes become more pronounced with considerable pain, stiffness, numbness, tingling, coldness, livid discoloration, and inability to either fully

bony proliferation occurs It is presumed, that in the presence of a myocardial infarction, pain impulses originating in the heart pass up sensory fibers to the "internuncial pool" of the lower cervical-upper dorsal cord and are relayed down the inside of the arm and side of the chest via the first, second, and third thoracic sympathetic rami. For a more complete discussion of the shoulder-hand syndrome see the chapter on the Painful Shoulder p. 971.

It has also been suggested that palmar contractures may originate as a ... remnants of the super- ... present in many of the ... of flexors Krogius has described islets of this muscle in the palmar aponeurosis of the newborn.[25]

Pathology.—Normally, the palmar fascia or aponeurosis is a triangular membrane consisting of four layers with its apex at the transverse carpal ligament. The superficial layer separates into four bundles one of which goes to each of the fingers; the thumb may receive a tiny band.[25] They attach to the deeper layers of the skin proximal to the webs and extend into the bases of the fingers. Some fibers run vertically and obliquely binding the skin to the aponeurosis, and giving rise at the level of the metacarpal heads to creases Transverse fasciculi communicate beneath with the three deeper fascial layers, the metacarpal bones, phalanges, and interphalangeal joints.[20,21,22] The palmar aponeurosis serves as a protection for the important compartments of the hand and helps to stabilize the skin so that it does not slide loosely on its base when the hand is used.

Early changes *consist of small, thickened plaques* at the level of the distal palmar crease overlying the tendons to the fourth and fifth fingers. The index and middle fingers may be subsequently involved, and rarely the thumb. *Microscopic sections of plaques* show thickening of the corneal ... replacement of normal ... the connective tissue ... mary feature by earlier writers. ... Others have thought ... l process starts in the interstitial connective tissue with associated inflammatory signs in the fibroblasts about the capillaries, in the skin, and interstitial connective ... he essential feature ... [16,17] Disruptions in ... that the trauma of ... vicious cycle

str ... in

ha ... han ... the

the left in unilateral disease. Males are pre...

incidence rises sharply in the fourth decade with little levelling off through the seventh The ring finger is most frequently involved, the little, middle and index fingers follow in that order When several fingers are affected, the involvement may not occur at the same time and may not progress at the same rate [44] [illegible]
disability or local sensitivity,
routine physical examination
[illegible]

Diffuse thickening over the entire volar aspect of the hand may appear [51]

Diagnosis is made by visual inspection and palpation Congenital or spastic contractures or those following injury or infection can be differentiated by having the patient flex his wrist, this maneuver relieves shortening of the flexor tendons and permits full extension of the fingers In Dupuytren's contracture extension of the fingers is not possible after bending the wrist When associated with the shoulder-hand syndrome, the hands may at times resemble those seen in scleroderma and Raynaud's disease.

Signs of Dupuytren's Contracture

Development of a *nodule on the palm of the hand* or fingers
Most commonly affected region palmar aspect of the metacarpophalangeal joint of the ring finger
Puckering of the palmar skin, most frequently at the base of the fourth and fifth fingers
Further nodules and funnel-like depressions of the palm occur with more induration and creasing of the skin and atrophy of the subcutaneous tissues
[illegible]
but this is very uncommon
Progressive involvement of the skin and fingers
Gradual and varying degrees of *contractures of the fingers*
Limitation of motion of the fingers to varying degrees by the contracted fascial bands
Months or years later [illegible]
[illegible]
of the fingers

Treatment.—An initial period of weeks or months should be devoted to *intensive physical therapy* unless the contracture is of long standing and claw fingers are present In both rheumatoid and degenerative arthritis minor degrees of this contracture occur rather frequently Conservative management relies on the use of heat, massage and stretching, exercises, splints and traction, galvanism and electrical stimulation The whirlpool bath is frequently employed, and the patient instructed to carry out active

stretching of the fingers while in water. For use at home the [illegible]
or [illegible]
pos[illegible]
ext[illegible] Home treatment should be
performed several times daily. *Deep finger kneading massage* is a sine qua non following heat The therapist or trained relative should fix the patient's hand with the fingers in as full extension as possible and carry out deep friction kneading with his free fingers. This should be done several times weekly, or preferably every day at home *Extension exercises* are then undertaken for ten minutes, and a splint applied[1,5,6,35] to maintain the advantage gained The splint can be worn overnight. A finger exerciser can be improvised out of one or two obstetrical type scales[41] Electrical stimulation may be used when vigorous contractions are desired Negative galvanism employing a cloth soaked in salt solution under the active electrode produces considerable softening of the fibrous bands, after such treatment for 15-60 minutes the therapist may be able to obtain full extension by stretching. If surgery is necessary, the post-operative program follows the same pattern.

In those cases associated with the shoulder-hand syndrome intensive physical therapy is possible *by concurrent use of either ACTH or cortisone.*[3,7,47,49] Full functional rehabilitation can be secured in a relatively short time Re-establishment of muscle power and bulk will require a longer period. Exercises, preferably of the resistance type, should be carried out several times daily for months Except in the most stubborn cases cortisone need not be used for more than one to two weeks, and in smaller doses (50–75 mg. daily) than employed in rheumatoid arthritis, if relapse occurs, a subsequent course of the hormone can be instituted. If it becomes possible to increase the solubility of cortisone, percutaneous absorption by inunction may be feasible and appears theoretically ideal as a means of treatment because of its known effect on connective tissue. Hollander[15a] has recently treated three cases of Dupuytren's contracture by repeated infiltration of the involved fascia with a suspension of hydrocortisone (compound F), and has noted a gradual softening and improved function as a result

X-ray therapy may occasionally produce temporary arrest in the early phases of this condition, but is of no value after firm contracture has developed. Vitamin E has some enthusiastic adherents[39,45] but this has not been confirmed[53].

The only relief of advanced cases of Dupuytren's contracture is by *adequate surgery and good post-operative care.*[11,27,32,48,51] *Complete excision of the palmar aponeurosis is necessary.* Subcutaneous fasciotomy or division of the contracted bands may be done occasionally prior to more extensive surgery.[4] Operation should be done before advanced fibrosis of the skin develops since devitalization may occur after separation of the skin from the scar, skin grafting then becomes necessary, and the operative [illegible] to strip the fascia [illegible] ximal phalanges [illegible] as an aid to complete hemostasis, vessels and nerves are often caught in the contracted fascia, and separation from the encasing fascia is difficult. Three approaches are used. a single incision paralleling the distal palmar crease,[1,14,21]

a double incision, one following the distal palmar crease and the other curved along the thenar eminence,[10] a curvilinear or L-shaped incision, first paralleling the distal palmar crease and extending toward the wrist along the ulnar side of the palm.[4, 34]

The aponeurosis is separated throughout the palm from the skin and subcutaneous tissue while still under tension, and then divided from its proximal attachment. At the bifurcation of the tendinous bands, the septa become visible, forming partitions between the tendons and neurovascular bundles. Longitudinal incisions along these septa detach them from the deep palmar fascia.[41] Dissection is carried out beneath the distal skin flap, freeing the band-like extensions from the transverse metacarpal ligament. Any involved fingers are exposed at the proximal phalanges to permit linking with the palmar incision. The aponeurosis is then removed en bloc. Devitalized skin is removed if present and replaced by a full thickness skin graft from the medial aspect of the upper arm. Grafting has become less necessary due to improvements in technique. Rubber drains are inserted before closure, which may be removed the following day. Postoperative hematomata not infrequently develop in the potential space created by removal of the aponeurosis in spite of pressure dressings and good hemostasis. Tanzer has recently described *a compression suture*, which is double-armed and incorporates a button on the volar side, one suture is placed between the second and third metacarpal bones under direct vision so as to avoid the neurovascular bundle, the second suture is introduced between the fourth and fifth metacarpal bones. These are threaded through a second button on the dorsal surface. Packing is carefully tucked beneath and around the loop in the palm, and the dorsal ends tied over the button securely. A posterior plaster, acrylic, or aluminum[5, 33, 41] splint is applied over the dorsum, and immobilization is maintained for a week to ten days.

When the skin incision is well healed, whirlpool (water temperature at 102-100° F.) may be used once or twice daily for thirty minutes during which the patient's fingers may be passively extended. Assisted active exercise is then instituted, and later active flexions and extensions are carried out repeatedly throughout each day. The posterior extension splints are worn at night and during inactive daytime periods until good function can be demonstrated.

BIBLIOGRAPHY

1 ADAMS, H D Surg Clin North America, 22, 899, 1942
2 ASKEY, J M Am Heart Jour, 22, 1, 1941
3 BAXTER, H, JOHNSON, L, MADER, V, and SCHILLER, C Canad Med Assn Jour, 63, 540, 1950
4 BUNNELL, S "Surgery of the Hand," 2nd ed, Philadelphia, J B Lippincott Company, 1949
5 ——— Am Jour Occupat Therapy, 4, 145, 1950
6 BUNNELL, S, and HOWARD, L D, JR Jour Bone and Joint Surg, 32-A, 226, 1950
7. BOWIE, M A Unpublished observations
8 COENEN, H Ergebn d Chir u Orthop, 10, 1170, 1918
9 CHRISTMAN, L D, and BUEHLER, V L Phys Therapy Rev, 30, 327, 1950
10 DAVIS [illegible] rg, 24, 933, 1932
[illegible] Soc nat de chir, 58, 424, 1932

stretching of the fingers while in water. For use at home the washbowl or paraffin dips serve as a good substitute. With *paraffin*, exercise is not possible, but the softening effect on the contracted tissues permits greater extension when the casts are stripped off. Home treatment should be performed several times daily. *Deep finger kneading massage* is a sine qua non following heat. The therapist or trained relative should fix the patient's hand with the fingers in as full extension as possible and carry out deep friction kneading with his free fingers. This should be done several times weekly, or preferably every day at home. *Extension exercises* are then undertaken for ten minutes, and a splint applied[4,5,6,35] to maintain the advantage gained The splint can be worn overnight. A finger exerciser can be improvised out of one or two obstetrical type scales[41] Electrical stimulation may be used when vigorous contractions are desired. Negative galvanism employing a cloth soaked in salt solution under the active electrode produces considerable softening of the fibrous bands, after such treatment for 15–60 minutes the therapist may be able to obtain full extension by stretching. If surgery is necessary, the post-operative program follows the same pattern

In those cases associated with the shoulder-hand syndrome intensive physical therapy is possible *by concurrent use of either ACTH or cortisone.*[1,7,47,49] Full functional rehabilitation can be secured in a relatively short time Re-establishment of muscle power and bulk will require a longer period Exercises, preferably of the resistance type, should be carried out several times daily for months. Except in the most stubborn cases cortisone need not be used for more than one to two weeks, and in smaller doses (50–75 mg. daily) than employed in rheumatoid arthritis; if relapse occurs, a subsequent course of the hormone can be instituted. If it becomes possible to increase the solubility of cortisone, percutaneous absorption by inunction may be feasible and appears theoretically ideal as a means of treatment because of its known effect on connective tissue. Hollander[19a] has recently treated three cases of Dupuytren's contracture by repeated infiltration of the involved fascia with a suspension of hydrocortisone (compound F), and has noted a gradual softening and improved function as a result.

X-ray therapy may occasionally produce temporary arrest in the early phases of this condition, but is of no value after firm contracture has developed. Vitamin E has some enthusiastic adherents[19,45] but this has not been confirmed[24]

The only relief of advanced cases of Dupuytren's contracture is by *adequate surgery and good post-operative care.*[11,27,32,43,51] *Complete excision of the palmar aponeurosis is necessary* Subcutaneous fasciotomy or division of the contracted bands may be done occasionally prior to more extensive surgery.[4] Operation should be done before advanced fibrosis of the skin develops since devitalization may occur after separation of the skin from the scar, skin grafting then becomes necessary, and the operative [illegible] necessary to strip the fascia [illegible] the proximal phalanges. [illegible] ployed as an aid to com- [illegible] aught in the contracted [illegible] is difficult Three approaches are used: a single incision paralleling the distal palmar crease;[1,14,21]

a double incision, one following the distal palmar crease and the other [illegible] a curvilinear or L-shaped incision, [illegible] ease and extending toward the wrist [illegible]

[illegible] ughout the palm from the skin and subcutaneous tissue while still under tension, and then divided from its proximal attachment At the bifurcation of the tendinous bands, the septa become visible, forming partitions between the tendons and neurovascular bundles Longitudinal incisions along these septa detach them from the deep palmar fascia [51] Dissection is carried out beneath the distal skin flap, freeing the band-like extensions from the transverse metacarpal ligament. Any involved fingers are exposed at the proximal phalanges to permit linking with the palmar incision The aponeurosis is then removed en bloc. Devitalized skin is removed if present and replaced by a full thickness skin graft from the medial aspect of the upper arm Grafting has become less necessary due to improvements in technique Rubber drains are inserted before closure, which may be removed the following day. Postoperative hematomata not infrequently develop in the potential space created by removal of the aponeurosis in spite of pressure dressings and good hemostasis Tanzer has recently described *a compression suture*, which is double-armed and incorporates a button on the volar side, one suture is placed between the second and third metacarpal bones under direct vision so as to avoid the neurovascular bundle, the second suture is introduced between the fourth and fifth metacarpal bones These are threaded through a second button on the dorsal surface Packing is carefully tucked beneath and around the loop in the palm, and the dorsal ends tied over the button securely. A posterior plaster, acrylic, or aluminum[5 32 51] splint is applied over the dorsum, and immobilization is maintained for a week to ten days

When the skin incision is well healed, whirlpool (water temperature at 102-106° F) may be used once or twice daily for thirty minutes during which the patient's fingers may be passively extended Assisted active exercise is then instituted, and later active flexions and extensions are carried out repeatedly throughout each day The posterior extension splints are worn at night and during inactive daytime periods until good function can be demonstrated

BIBLIOGRAPHY

1 ADAMS, H D Surg Clin North America, 22, 899, 1942
2 ASKEY, J M Am Heart Jour, 22, 1, 1941
3 BAXTER, H, JOHNSON, L, MADER, V, and SCHILLER, C Canad Med Assn Jour, 63, 540, 1950
4 BUNNELL, S "Surgery of the Hand," 2nd ed, Philadelphia, J B Lippincott Company, 1949
5 ——— Am Jour Occupat Therapy, 4, 145, 1950
6 BUNNELL, S, and HOWARD, L. D, JR Jour Bone and Joint Surg, 32-A, 226, 1950
7 BOWIE, M A Unpublished observations
8 COENEN, H Ergebn d Chir u Orthop, 10, 1170, 1918
9 CHRISTMAN, L D, and BUEHLER, V L Phys Therapy Rev, 30, 327, 1950
10 DAVIS, J S [illegible] rg, 24, 933, 1932
[illegible] Soc nat de chir, 58, 424, 1932

14 Gill, A. B Ann. Surg, **77**, 221, 1919.
15 Hohmann, G. Ztschr f Orthop, **73**, 45, 1941.
15a Hollander, J L Personal communication.
16 Horwitz, T: Arch Surg, **44**, 687, 1942
17 Ikle, C Deutsch Ztschr. f Chir, **212**, 106, 1928
18 Janssen, P Arch f klin Chir, **67**, 761, 1902
19 Johnson, A C Ann Int Med, **19**, 433, 1943
20 Kalberg, W. Anat Anz, **81**, 149, 1935
21 Kanavel, A B, Koch, S L, and Mason, M L: Surg, Gynec and Obstet, **48**, 145, 1929
22. Kaplan, E B: Surgery, **4**, 415, 1949.
23 Kehl, K C: Ann Int Med, **19**, 213, 1943.
24 King, R A Jour Bone and Joint Surg, **31-B**, 443, 1949
25 Krogius, A Zentralbl f Chir, **47**, 914, 1920.
26 ———: Acta chir Scandinav, **54**, 33, 1921.
27 Lexer, E "Die gesamte Wiederherstellungschirurgie," 2nd ed, Leipzig, Johann Ambrosius Barth, 1931
28 Lund, M Acta psychiat et neurol, **16**, 465, 1941.
29 Manson, J S: Brit Med Jour, **2**, 11, 1931
30 Meyer, J C, and Binswanger, H F Am Heart Jour, **23**, 715, 1942
31. Meyerding, H W Proc. Staff Meetings, Mayo Clinic, **10**, 694, 1935
32 ———: Arch Surg, **32**, 320, 1936
33 ———. Am. Jour Surg, **49**, 94, 1940
34 Meyerding, H W., Black, J R., and Broders, A C: Surg, Gynec. and Obstet, **72**, 582, 1941
35 Meyerding, H. W, and Krusen, F H. Physiotherapy Rev., **16**, 42, 1936
36 Nichols, J B M. News, **75**, 491, 1899
37 Nierderlan, W Med Wlet, **7**, 126, 1933
38 Nippert: Deutsch Ztschr f Chir, **216**, 289, 1929
39 Parsons Irish Jour Med. Sci, 272-274, 1948
40. Powers, H. Jour. Nerv and Ment Dis, **80**, 386, 1934
41. Rudd, J L. Arch. Phys Med, **32**, 347, 1951.
[illegible] **25**, 125, 1934-35.

[illegible] in "Proceedings of [illegible] ed by J. R Mote,

50 Tanzer, R C, in Proc of Forum Sessions, American College of Surgeons, **2**, 210, 1951
51 Tanzer, R C New England Jour Med, **246**, 807, 1952

CHAPTER 56

PAINFUL FEET

Revised by John G. Kuhns, M.D., and Theodore A. Potter, M.D.

Painful feet are found very commonly in chronic arthritis.[33] The joints of the hands and feet are the joints most commonly involved in this disease.

ties of the feet respond readily to intelligent treatment.[53] Arthritis in the feet responds somewhat more slowly but in most instances a painless, useful foot can be obtained.

The disabilities of the feet can be divided for clinical convenience into the following groups:

1. Static Disabilities
   - (a) Weak feet
   - (b) Deformed feet
   - (c) Tight Tendo Achilles
2. Traumatic Disabilities
   - (a) Acute Sprain
   - (b) Fractures and dislocations
3. Inflammations
   - (a) Of bones
   - (b) Of joints
   - (c) Of soft tissues
4. Congenital Disabilities
   - (a) Relaxed foot
   - (b) Club foot
   - (c) Long second metatarsal
5. Developmental Disabilities
   - (a) Lessened growth of leg or foot
   - (b) Epiphyseal disturbances
6. Circulatory Disabilities
   - (a) Arterial
   - (b) Venous
   - (c) Neurovascular

Over 90 per cent of painful feet are caused by static disabilities. These

genital disabilities usually come under the care of orthopedic surgeons early

there are symptoms. *Circulatory disturbances are most commonly found in the aged.* While most of these can be helped, few of them can be cured

disease. Pain in the sole of the foot may be caused also by deformities,

injuries, neoplasms, *contractions; scar tissue;* absorption of fat from pressure or after prolonged illness, and plantar neuralgia

Predisposing Causes of Foot Strain
Congenital relaxation of ligaments
Disturbances in weight carriage—genu valgum, coxa vara, dislocation *of the hip*
Poor posture
Badly fitting shoes and stockings (especially *short shoes and stockings*)
Prolonged illness (prolonged bed rest)
Obesity
Occupations requiring prolonged standing and walking
Circulatory impairment
Systemic diseases which produce weakness or malnutrition

Circulatory disorders of the foot which may present cutaneous manifestations include: obliterating arteriosclerosis, thrombo-angiitis obliterans, acrocyanosis, Raynaud's syndrome, erythromelalgia, *immersion foot* and vascular anomalies (such as port wine or capillary angioma, spider nevus, small cavernous angiomas, lymphangiomas and diffuse lymphangiomatosis of the leg and foot)

NORMAL PHYSIOLOGY OF THE FEET

The center of gravity of the body is normally balanced over the triangular weight-bearing area of the foot, with its apex posteriorly. This weight is divided equally between the heads of the metatarsal bones and the tuberosity of the os calcis. The anterior shift of the center of gravity which accompanies poor posture disturbs the muscle balance in the foot, and causes a strain on one group of these, leading to what is known as the weak foot.[64] Physicians desiring detailed anatomic or physiologic data on this *subject are referred to the works of Dickson and Dively, Morton,* Lake, Lewin, and Hauser.

The foot is in part like a spring, composed of a bony framework with

ing leather covering which may compress and deform the forefoot and weaken its muscles. Inasmuch as the shape of the shoe very seldom corresponds to the shape of the adult foot, the shoe is one of the most important factors contributing to foot strain

Physicians commonly speak of a *longitudinal* and a *transverse arch* of each foot. The longitudinal arch runs anteroposteriorly along the inner border of the foot from the tuberosity of the os calcis to the heads of the metatarsals, centering about the inner three metatarsals, cuneiforms, scaphoid, astragalus and the calcaneus. The medial portion of the longitudinal arch rests upon the os calcis and the head of the first metatarsal.[72] The anterior transverse metatarsal arch passes through the necks of the

. . . . of the first and fifth metatarsals

... the weight ... he ground in weight bearing so that no transverse arch is present at that ime.

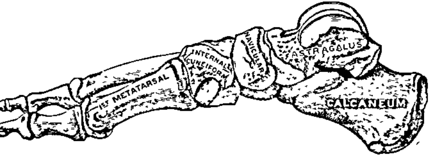


Fig 311.—The bones of the right foot, viewed from the inner side (Gerrish's Anatomy)

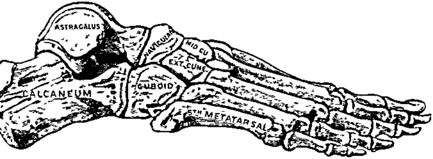


Fig 312 —The bones of the right foot, viewed from the outer side (Gerrish's Anatomy)

arch as one approaches the base of the metatarsal bones When the feet are brought together the arches of both feet resemble a shallow, inverted bowl.

... the integrity of the longitudinal ... which is ... and short ... and outer part)

The strength and importance of the plantar fascia is demonstrated in paralysis of the extensor muscles of the foot A pes cavus develops with extreme contracture of the plantar fascia

The muscles are important structures in the foot The plantar flexors are approximately 5 times as strong as the dorsal flexors and the gastrocnemius and soleus are 3 times as powerful as the remainder of the foot

muscles combined.[51] The chief movements of the foot are carried out by the tibial and peroneal muscles. The tibialis anterior elevates and the peroneus longus depresses the first metatarsal. The tibialis posterior produces inversion and adduction. The peroneal muscles aid eversion and abduction.[50] The chief evertors of the foot are the peroneus longus and peroneus brevis. The invertors are chiefly the tibialis anterior and tibialis posterior. The normal ratio of strength of the invertors to the evertors should be as 6:5.

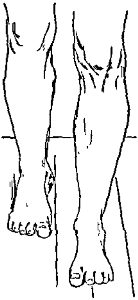


FIG 313 —Illustrating the automatic adduction of the forefoot on the right side, due to the obliquity of the bearing surface of the metatarsus, in the proper attitude for walking (Whitman)

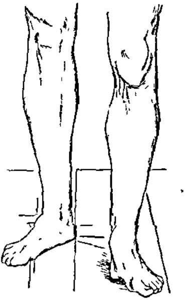


FIG. 314 —The improper attitude of outward rotation of the limbs usually accompanied by eversion of the feet, in which there is disuse of the leverage function. (Whitman)

Definitions of Positions of Foot

Adduction Forefoot displaced inward in relation to midline of limb
Abduction: Forefoot displaced outward in relation to midline of limb
Inversion Sole of foot turned inward
Eversion: Sole of foot turned outward
Adduction and inversion and abduction and eversion are often combined
Valgus. Deformity of the foot in abduction and eversion
Varus Deformity of the foot in adduction and inversion
Calcaneus Deformity of the foot in dorsiflexion
Equinus Deformity of the foot in plantar flexion
Supination Same as inversion.
Pronation: Same as eversion.



Fig 315 —Distribution of weight to the foot (Geo E Keith Company)



Fig 316 —Weight-bearing points of the foot (Geo E Keith Company)

Examination of the Foot

History of pain or preexisting arthritis Relation of pain to standing and walking Pain while resting Stiffness on arising Relation to work

Foot strain may be associated with (1) Pregnancy with gain in weight or muscular relaxation (2) Obesity—increased load upon the feet (3) Backaches which may aggravate foot strain or may be caused by foot strain (4) Injuries to the leg or foot (5) Change of occupation or greater activity in the occupation requiring more walking or standing (6) Changes in type of footwear—from high to low heels, from stiff to flexible soles

History should include duration and course of the disability and the type and result of treatment

The gait should be observed

See that the patient's shoe is not too short, long, narrow or wide Note the points of greatest wear on the shoe With short tendo Achilles the tips of the soles are scuffed and worn In simple eversion the [illegible] outer side of the [illegible]sal bones a hole [illegible] part of the foot [illegible]he shoe

Examination of the Foot—*(Continued)*

Examine the posture of the body particularly *excessive lordosis* and flexion and rotation at the hip and flexion or genu valgum at the knee Are the legs of equal length?

Examine the foot at rest and on weight bearing Look for prominences on the inner side of foot, spread of the forefoot, shape and position of toes

See if the heel is held in varus or valgus.

Look for corns and calluses, prominence of metatarsal heads on sole of foot, bony prominences at side or dorsum of the foot, swelling about the malleoli, thickening over any tendon, plantar warts, varicose veins, ulceration, blueness or coldness of the feet.

Palpate the joints for swelling or tenderness

Look for tenderness under the calcaneal fat pad, in the plantar fascia, the inner and outer side of the ankle

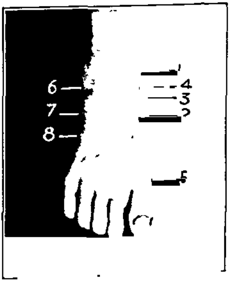
See to what extent the longitudinal and transverse arches are depressed with weight bearing.

[illegible] If the [illegible]

for epidermophytosis between the toes

Palpate the dorsalis pedis and posterior tibial arteries.

X-ray examination is indicated if any gross variation from the normal is found



[illegible] of the

FLAT (WEAK) FEET

a better name for this type of flat foot might be abducted or weak foot because some of these patients may have high arches and may suffer greatly. During activity these individuals turn their feet outward so that the weight-bearing line prolonged downward through the center of the patella along the crest of the tibia no longer falls in the normal position between the first and second toes, but may be to the inner side of the first metatarsal.[24]

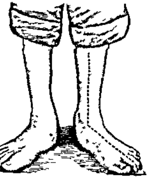


Fig 318



Fig 319

Fig 318 —Typical ... t ele-
m ... promi-
ne ...

... occur,
... eroneal

The rigid flat foot is an end stage, with the foot firmly fixed in abduction by contractures and adhesions in which simple measures will not restore the normal balance of the foot. Many of these patients have severe pain

The causes of weak feet are legion but they can be grouped into congenital, developmental, occupational, systemic, traumatic and neurological categories. In those seen in early childhood there is usually a congenital relaxation or weakness of muscles and ligaments. In the developmental group in addition to faulty posture, abnormal development in tarsal or

to weakening, distortion and limitation of motion in these structures

Trauma may include a fracture of the os calcis, sprained ankle, Pott's fracture, short leg, favoring one foot after injury or because of a wart on the sole, Morton's toe, corns, etc.

Vocational flat feet may develop from standing on hard surfaces for long periods, as in policemen, mailmen, physicians, dentists, waiters, teachers, barbers and bakers.

Failure of the tibialis anterior and posterior (which maintain the longitudinal arch of the foot) throws strain upon and stretches the ligaments

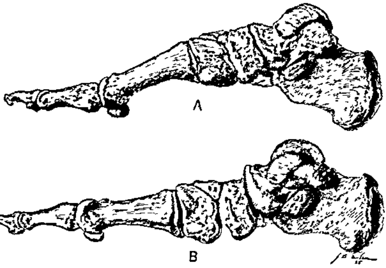


FIG 320.—Medial aspect of the foot *A*, Normal longitudinal arch *B*, Arch in flat foot (Shands, Orthopedic Surgery, courtesy of the C V Mosby Company)



FIG. 321.—The ordinary type of weak foot in a child The degree of abduction indicated by the relation of the patellæ to the feet (Whitman)

with weight bearing, this leads to the pain in early flat foot Foot strain is common in adolescence and early adult life, particularly in those who stand for long periods. If these individuals are treated as soon as pain or tiredness of the feet is noted, one may prevent the development of a deformity.

Flat feet are caused mainly by improper shoes, overstrain and debilitating illness Foot strain may be alleviated by proper shoes and snugly fitting supports for weak feet or strained arches but foot exercises and training in correct walking are required to develop the foot muscles and relieve symptoms entirely.

lar fatigue and weakness. It is only after both the muscles and ligaments give way that flat feet occur.

Strain of the anterior arches is due mainly to cramping of the forefoot, this will usually not occur if one wears shoes which are wide enough, and properly fitting stockings; treatment includes raising the arch by pads, or by a metatarsal bar, and by exercises

Morton[18] states that the commonest pathologic causes of pronated feet are: Abnormal laxity of the plantar ligaments which permits excessive dorsal mobility so that the first metatarsal does not come into firm contact with the ground at the same time as the other metatarsals, and a congenitally shortened first metatarsal bone

Symptoms of Weak Painful Feet

aspect of the head of the first metatarsal bone, occasionally with numbness of the toe and pain near the origin of the plantar fascia in the heel and at or under the external malleolus, in weak feet

One may have pain in the feet, legs and low back for hours on retiring, this usually disappears by morning

Burning or callus formation on the soles of the feet under the heads (distal

the feet

In both of these conditions, weight which is borne normally by the first metatarsal segment is shifted at least in part to the second This latter bone is not built to support such added strain and its ligaments become

Signs of Painful Flat [illegible]

to the great toe.

Prominence of the internal malleoli with a prominent convex curve along the inner and inferior aspects of the arch.

Pain and tenderness beneath the scaphoid, head of the astragalus, sustentaculum tali and internal malleolus and along the plantar surface of the inner border of the foot and over the central portion of the plantar fascia.

Lengthening of the inner borders of the feet (because of separation of the os calcis and scaphoid, stretching of the spring ligament, and dropping of the head of the astragalus between the two bones).

A downward and outward position of the tendo Achilles (instead of the normal directly downward position)

Swelling of the feet, especially anterior to the internal and external malleoli List of the foot to the inner side, leg seems displaced medially.

Marked wear of the soles of the shoes along the inner side. Weight is borne on the inner side of the foot.

Knock knee may be an early sign of development of flat foot

In weak feet, with peroneal spasm, there is slight or marked limitation to adduction or turning in of the foot when the leg is extended. The flat foot is actually a valgus foot, *i e.,* abducted and everted.

Secondary symptoms include *tenderness and callus formation under the first metatarsophalangeal joint* and the inner side of the heel, callus formation beneath the prominent scaphoid bone (if marked relaxation of the longitudinal arch occurs), and an awkward gait

If not relieved, eversion and abduction lead to osseous, irreducible flat foot When rigid flat foot has occurred, there is no spring in the gait and patients use their lower extremities like pedestals.

Deformities secondary to flat feet include hallux valgus, hallux rigidus and hammer toe

Roentgen examinations of the feet may aid in establishing an accurate diagnosis in some cases, this includes a dorsoplantar roentgen-ray film centering upon the middle cuneiform bone. A lateral view is of value in lesions of the posterior part of the foot, and an oblique view may be needed for the mid-tarsal region. Findings of importance in such films include

- Enlargement of the second metatarsal bone (in comparison to the three outer metatarsals)
- *Shortness of the first metatarsal,* noting the position of its head in comparison to the head of the second metatarsal
- Partial union of astragalus and os calcis
- Navicular-cuneiform sag
- Supernumerary bones may be present especially an accessory scaphoid.
- Loss of cartilage and development of bony ridges and deformities in the intertarsal and metatarsophalangeal joints in most cases

degrees), so that strain falls upon the ligaments Increased strain falls upon the inner side of the foot when the feet are turned out in walking.

In the strained foot there is overstretching of the inferior calcaneoscaphoid ligament, plantar fascia and plantar ligaments and straining of the muscles controlling the inner side of the foot.[12] Relaxation of the calcaneoscaphoid ligament permits forward pressure of the head of the astragalus. The latter approaches and finally crosses the inner border of the foot print. As a compensatory mechanism, abduction, dorsiflexion and supination occur in the forepart of the foot.

With this there follows an internal rotation of the knees and of the hips with a resulting forward tilting of the upper portion of the pelvis This produces a lumbar lordosis when the shoulders are brought backward in order to maintain a state of equilibrium Thus, foot strain is a very common cause of backache.

Standing with the foot rotated outward produces muscular fatigue and if persisted in for long periods stretches the ligaments on the inner side of the foot and produces weak foot. If the forefeet are rotated outward in walking, the ligaments and muscles on the inner side of the foot are stretched and weakened as most of the weight is thrown on this area, again leading to flat foot.

Outline of Treatment in Early, Flaccid Flat (Weak) Foot
No weight bearing if pain is present at rest *Proper shoes, with heel and sole wedges,* if indicated [illegible] Reduction of weight if indicated Night plaster boot with ankle at right angle and foot in slight inversion if there is persistent muscular spasm

Treatment of the Weak (Flat) Foot.—Pain and impaired function are indications for treatment. If pain is present at rest or if there is an associated arthritis, prohibit weight bearing until pain subsides if this is

[illegible]

muscles of the foot; exercises must not cause muscular fatigue or pain.

Many individuals with weak, pronated, painful feet may be markedly benefited by a longitudinal arch pad of felt, cork or rubber, averaging $\frac{3}{16}$ inch (beveled to $\frac{1}{32}$ inch on the lateral side) and a heel wedge at the rand (just beneath the sole). The proper placing of the rand wedge is extremely important, lest one turn the heel of the shoe and not the foot.

The physician must insist that the entire heel be taken off, the wedge placed next to the sole (heel rand) and that the heel then be replaced. This wedge averages $\frac{1}{4}$ inch in thickness with its highest point at the breast of the heel (anteriorly on the mesial aspect), running to $\frac{1}{32}$ inch posteriorly and laterally.[27]

In mild cases or those associated with knock knee, throw the weight toward the outer side of
by raising the inner bor
mately $\frac{1}{4}$ inch. Shaping
foot in weight bearing may prove useful.

In very flexible weak feet the patient often obtains greater comfort with the use of a pronatory inner wedge under the heel and a supinatory outer wedge under the little toe. These are not used in rigid or in juvenile flat foot. The wedge raises the inner border of the heel so that the os calcis is

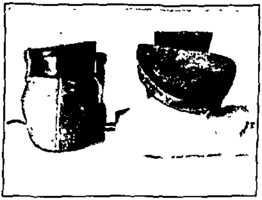


FIG 322 —Shoes corrected with cleats; inner wedge heel and outer patch over toes (Steindler, Jour Bone and Joint Surg)

placed squarely on the ground. In addition, in order to equalize any
heel
the
urity

In patients with short Achilles tendons, the whole heel must be raised temporarily to relieve this strain.

If necessary, an arch support is used temporarily when the patient

exercise without fatigue or pain in the feet.
and in elderly individuals the supports must often be used permanently.

Arch Supports.—These are very useful appliances in the relief of the symptoms from weak feet. In general two types are used; tempered steel plates and leather inlays The steel plates are either coated with a rust-resisting coating or covered with leather. These plates are molded to

support the longitudinal and transverse arches They are most satisfactory when made from plaster impressions of the non-weightbearing foot. About one-half of those which are used are manufactured in a small number of standard sizes. While these can be obtained readily from surgical supply houses, they rarely fit accurately and do not give as much relief as those which are made from plaster impressions of the patient's foot

Leather inlays fit in the shoe like insoles. In these the arches are supported by molded areas of felt, sponge rubber or cork which are incorporated in the inlays under the arches of the foot Both of these types of arch supports have the advantage that they can be adjusted to fit the changing need of support as the foot becomes less strained and fatigued In this they are superior to any rigid support.[27]

[illegible]

Usually steel plates are better for most adults and in feet that do not come



FIG 323 —Type of metal foot plate sometimes used to support the arches (Kuhns, courtesy of Rheumatism)

into varus readily. In children and in those with circulatory disturbances relatively soft leather inlays are more comfortable and satisfactory in most cases. Generally, the foot supports give only slight pressure under the arches of the feet at first. As symptoms subside and the foot becomes more flexible, more pressure is exerted In plates this is done by pounding or bending up the raised portion of the steel plate further At the same time an attempt is made to hold the foot in slight varus and slight adduction In leather insoles or "foot pads" more felt or rubber can be put under the arches until symptoms are completely relieved and the foot is held in a go[illegible]
are being [illegible]
In the un[illegible]
number o[illegible] and are gradually given up as normal strength and alignment return to the foot. In older patients and in those whose feet can not be made normal arch supports may be required all of the patient's life

In the milder forms of foot strain pads cut from felt or firm sponge rubber can be fitted into the shoe under the arches of the foot and held in place with glue or rubber cement

If the patient can rest for days or weeks with practically no weight bearing, to relieve strain, and then is given exercises and proper footwear,

often no arch support may be required. But few individuals can rest for a

of

m

a

su

extend only to the fifth metatarsal bone. The height should conform in

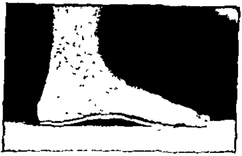


FIG 324 —Metal plate beneath the foot. (Kuhns, courtesy of Rheumatism)

Use of Arch Supports in the Uncomplicated Weak Foot

Arch supports are a temporary measure to relieve strain and aid proper posture of the foot until fatigue subsides and the muscles of the foot again become strong and efficient with exercises.

Arch supports relieve but do not cure.

Arch supports assist the hammock function of invertor and evertor muscles and calcaneoscaphoid ligament. They should conform to the *shape of the arch and exert only slight pressure.*

The arches transmit stresses to the heel and to the metatarsal heads These stresses are greater in high-arched feet.

In general an arch support should be constructed from a plaster mold or a paper tracing of the patient's foot without weight bearing

... to a

... nner

... ne

... ports

should be modified during convalescence to conform to the changing shape of the foot. *This is done by raising the support as the muscles* and ligaments resume their function.

Supports are gradually laid aside when symptoms have disappeared and the patient has learned to use the foot in a normal manner

both longitudinal and transverse arches to the height of these regions when no weight is borne.

Indications for Longitudinal Arch Supports.—Individuals (especially if

change from a sedentary to an active

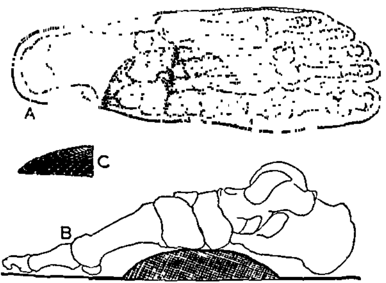


FIG 325 —Longitudinal arch support suggested by Shands *A*, Plantar view, *B*, medial aspect of the foot with the support in place; *C*, cross-section of the support showing the thickest portion of the medial aspect, tapering laterally (Shands, Orthopedic Surgery, courtesy of the C. V Mosby Company)

anterior d callus formation beneath the

the main disturbance in disorders of the longitudinal arch and of the anterior portion of the foot is a "functional deficiency of the first metatarsal segment," so that an excessive load and strain is placed upon the second

metatarsal segment and its tarsal joints (part of which is normally borne by the *first metatarsal segment*). Whether this is caused by laxity of the plantar ligaments or by a short first metatarsal bone, the point of strain is focused upon the basal joints of the second metatarsal. To relieve this disability an arrangement must be worked out which will permit the first metatarsal segment to again carry its normal portion of the body load.

If the disturbance is caused by a laxity of the plantar ligaments, a dorsal hypermobility results which prevents the first metatarsal from coming into firm contact with the ground at the same time as the other metatarsals, thus increasing the load borne upon the second metatarsal. Morton compensates for this condition by placing a support of sufficient height *under the head of the first metatarsal* so that contact is made by this metatarsal at the same time as this occurs with the other metatarsals, thus the first metatarsal segment again carries its portion of the burden, and allows the second metatarsal segment to support only its normal share of the body weight. For this purpose, there has been devised a compensating insole *which fits into the shoe with a "platform-like projection" just under the* head of the first metatarsal bone. This may also be useful in cases of foot strain due to a congenitally short first metatarsal bone. The height of the small platform thus placed can be determined only by trial, as the "compensating insole" can be removed easily for adjustment of the height of this *portion. In some cases however, any support under the first metatarsal* head causes mid-tarsal pain and tends to throw weight to the outer aspect of the foot causing irritation of the fifth metatarsal.

PROPER SHOES

Well fitting shoes of proper construction are necessary for walking under present-day conditions.[8] Soft moccasins or boots were adequate for warmth and protection when man walked on soft yielding earth, but feet will not withstand prolonged use upon hard pavements and floors without adequate protection. Many shoes now *being worn do not protect the feet in the* present unnatural environment in which most of us must live

Faults in choosing shoes fall into two categories: buying too short or small a shoe is probably the chief fault, and accepting a shoe that is not properly made for walking is the second fault. In both of these faults women err much more often than men—buying shoes that look nice rather than shoes that fit the foot properly Many women have the impression that shoes which are best for the foot are necessarily non-stylish and ugly.

The proper shoe should fit *If shoes are persistently uncomfortable they* should not be worn regardless of their quality or beauty. New shoes can be secured readily but foot disabilities can not be discarded readily and their correction often requires long time and effort. Shoes are now made over a relatively large number of standard lasts. There are few feet which *cannot be fitted properly* in ready made shoes if a few general instructions followed

determining the *culiarities of foot* s is also of value

Fitting of a foot requires careful measurement from heel to ball and from heel to toe This is essential for support and for function of the muscles on the inner side of the foot. Cramping of the toes leads to weakness and is soon followed by depression of the transverse arch. Insufficient space in the mid-foot may weaken and depress the longitudinal arch. Insufficient cuboid space does not give

tion where the one shoe is too large will often lead to a comfortable fit. For those with a large variation in the size of the feet, many large shoe stores will fit mismated shoes for each foot Those with very severe deformity can often only be fitted adequately by a maker of orthopedic shoes. These men can also make adjustments in the shoe for fixed deformities of the heel, forefoot or toes



FIG 326 —Compensating insole with platform to lie under head of short first metatarsal bone (Morton, The Foot, courtesy of Columbia University Press)

The disturbances in gait which are caused by improperly fitting shoes can be demonstrated by slow motion pictures, or by an ingenious electrical apparatus known as the electrobasograph [63] By this apparatus, Schwartz and his co-workers showed the increasing instability of the feet with increased height of the heel of the shoe They demonstrated incidentally that the stability of the foot was not related to the length, width and comfort of the shoe. They found that instability and abnormality in weight bearing was the result of improper design of the last It has been found that a correct shoe must contain sufficient space for independent movement of all the toes and should conform to the shape of the sole of the foot The foot and shoe should move as one

In order to fit the shoe properly and to order modifications intelligently one must know something about its manufacture and the various parts of a shoe. The *last* of a shoe is a wooden model similar to the shape of the foot,

either soft or firm ... to the laces ... The waist is ... This part is often too tight in high-arched feet. The shank is a thin supportive bar, usually steel, placed above the sole running from the anterior third of the heel forward almost to the metatarsal heads

For city wear ... more e... with g... or defo...

counter of the shoe is the heavy leather support around the back of the

shoe. This should fit snugly about the os calcis. This is essential if any arch support is worn.[10]

Shoe Corrections.—The shoe correction most frequently prescribed is a raise in the front inner corner of the heel $\frac{1}{8}$ to $\frac{3}{8}$ of an inch. This is indicated in mild valgus of the os calcis. It is often combined, especially in children, with relaxed feet, with arch supports. Usually in children the raise varies

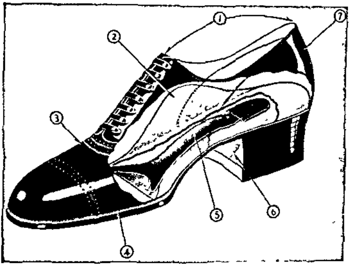


Fig. 327 —Shoe showing *shank* (5) of a shoe extending from the heel to the forepart of the foot. (Geo. E. Keith Company.)

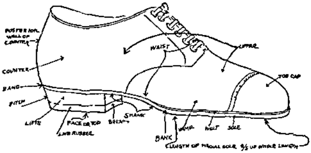


Fig 328 —External parts of a shoe (Ashley, courtesy of Medical Record)

between $\frac{1}{8}$ and $\frac{1}{4}$ inch. In adults usually $\frac{1}{4}$ inch is prescribed. Such raises are usually placed just beneath a rubber heel but may be put inside the shoe beneath the insole A *Thomas heel*, often combined with a raise of the front inner corner of the heel, is a forward extension of the heel on the

shoe transversely just behind the metatarsal heads. It is usually $\frac{1}{8}$ to $\frac{1}{4}$ of

an inch in thickness and about ⅜ of an inch wide. It is used chiefly in rigid depressions of the transverse arch associated with contracture of the extensor tendons and hammer toe deformity. It shifts the weight back to the bar from the metatarsal heads and usually affords comfort from severe anterior arch strain.[21]

A *cuboid wedge* is a raise ⅛ to ⅜ of an inch placed under the cuboid region on the lateral side of the sole. It is used to aid the correction of an adduction of the forefoot. It is of much value only in feet that are flexible

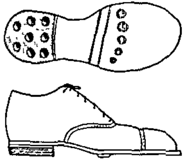


FIG 329

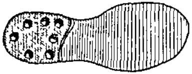


FIG 330

FIG. 329 —Metatarsal bar. (Shands, Orthopedic Surgery, courtesy of the C V Mosby Company.)

FIG 330 —Thomas heel; note the forward extension of the heel along the inner margin (Shands, Orthopedic Surgery, courtesy of the C V Mosby Company)

boots) or low shoes (oxfords). *Corrections will help the foot* ...ctions on shoes or firm corrective shoes are worn until symptoms subside and examination shows disappearance of the weakness or deformity. In young people this often occurs within six months. In older people they must sometimes be worn indefinitely.

Advantages of Orthopedic (Corrective) Shoes
Orthopedic shoes are built *heavier* than ordinary shoes The *shank is usually long and broad* (to allow enough base), is often triple fluted, and is placed in a definite position, usually on the outer central side of the sole, running from the heel to the beginning of the metatarsal bones Most orthopedic shoes have *long counters* which act as a base for the correction which is placed in the shoe, and give further support against pronation

Advantages of Orthopedic (Corrective) Shoes—(*Continued*)
The heel of an orthopedic shoe is *not higher than 1½ inches*, and usually has a broad base. (A high-heeled shoe pitches the body forward and would cause the patient to fall except for a compensatory lordosis as an attempt to regain equilibrium, there occurs an associated flexion of the knees and extension of the leg on the foot. This leads to back-strain, some displacement of the abdominal viscera, and shortening of the gastrocnemius muscle and the tendo Achilles These individuals then experience difficulty on attempting to change from high to low heels. *High heels lead to frequent sprains of the ankle because of the unstable base of a high, narrow heel.*
The orthopedic shoe has *greater depth in its throat to allow for the insertion of corrections* (such as arch supports) without binding the foot in the vamp
Orthopedic shoes are made with a *combination last* to insure a snug fit, and a well fitting heel which does not permit extra motion. They have a straight inner border which permits the great toe to lie in a natural position.



FIG 331 —*Incorrect walking position* (toes pointing outward) (Geo E. Keith Company)



FIG 332.—*Correct walking position* (toes pointing straight ahead). (Geo E Keith Company)

Proper Standing and Walking Positions[22]

Feet 4 inches apart
Toes pointing straight ahead
Weight on the outer margins of the feet.
Arches cupped as the palm of the hand
Head up, abdomen in
To demonstrate good posture, Stand with the back against a wall, with the head, shoulders, buttocks touching the wall and the heels slightly forward Place the hand between the small of the back and the wall Abnormal lumbar lordosis produces a large space here Flatten the lumbar spine by pushing the small of the back against the hand
Do not keep one position for a long period of time

Exercises and Postural Aids for Weak, Painful Feet[27,28]

Exercise is more important than mechanical supports in the treatment of weak feet Exercises strengthen the lifting muscles on the medial border and transverse portion of the arch (i e , the tibialis anterior and posterior, flexor longus hallucis, flexor longus digitorum, interossei and peroneus longus) Proper function of the foot muscles prevents painful lowering of the arch and renders mechanical support unnecessary Exercises must not be instituted in the acute stages of foot strain as they may aggravate the condition.

[illegible]

frequently as normal strength returns.

Exercises and Postural Aids for Painful Feet—(*Continued*)

1. Spread and flex the toes sharply downward; this may be done with the shoes off or on.
2. Stand on the toes with these turned inward, roll out on the sides *and return to a flat position.*
3. Place about 10 small marbles on the floor While sitting with the bare feet pick up 1 marble at a time with the foot and place it

4. Walk on the outer borders of the bare feet along a straight line
5. Stand with the toes pointing inward and attempt to externally rotate the knees without lifting the big toe from the floor.
6. While seated on the floor, bring one sole into contact with the other as much as possible.
7. Sit with feet pointing forward, curl all toes slowly, relax.
8. Sit squarely, roll feet outward until soles are almost parallel, relax.

from the wall, sway toward the wall keeping heels on floor. Do not bend at the knees.

If it is necessary to stand for long periods, rise on the toes and put weight on the outer border of feet, about every ten minutes

Children should be encouraged to walk in bare feet on sand or grass.[63]



FIG. 333.—The tip-toe exercise, raising the body on the adducted feet. (Whitman.)



FIG. 334.—The tip-toe exercise resting on the outer borders of the feet. (See Fig 333). (Whitman.)



Fig 335 —Usual exercise for stretching a contracted or tight tendo Achilles The heels should remain on the floor and the knees should not bend during the exercise

ADHESIVE STRAPPING TO THE WEAK FOOT

Pain may be [illegible]

In addition, the inner border of the heel may be raised about $\frac{1}{4}$ inch to relieve strain on the dressing and to get the patient used to carrying weight on the outside of the foot.

A simple form of adhesive dressing may be applied as follows (1) Paint [illegible]

are reached. Leave 1 inch free from adhesive on the dorsum of the foot to prevent edema. (6) Gauze is applied around the adhesive to hold this firmly to the skin and to prevent damage to the stocking. The gauze may be removed in twenty-four hours. Do not apply adhesive if there is marked eczema, many thin varicose veins or skin sensitivity.

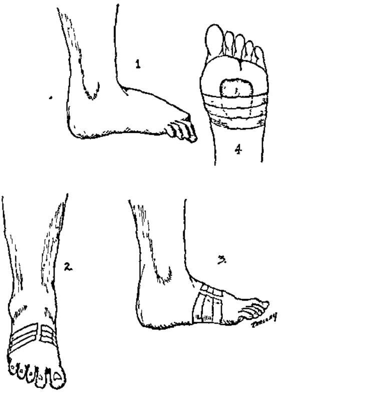


FIG. 336.—Anterior (metatarsal arch) strapping. Soft foot pad (as in 4) may be incorporated into strapping. Distal border of the pad must be proximal to the metatarsophalangeal joints. The pad lies over the second, third and fourth (but not the first or fifth) metatarsals. (Dickson and Diveley, Functional Disorders of the Foot, courtesy of J. B. Lippincott Company.)

Additional Aids in the Treatment of Painful (Weak) Feet Include: (1) Elastic or leather metatarsal strips with a metatarsal pad for patients with metatarsalgia; (2) the elastic web band where there is effusion in the midtarsal joints causing pain along the branches of the median plantar nerve; and (3) sponge rubber heel inserts in patients with tenderness of the tuberosity of the os calcis. (4) A figure-of-8 leather strap about the ankle is helpful for a weak ankle.

Physical therapy in the weak foot aids in breaking up adhesions and re-establishing a normal circulation. A warm whirlpool bath at a temperature of 100° F. for twenty minutes daily is helpful when edema and poor circulation are present. Massage of the muscles of the leg and foot, manipulation of the foot and ankle joints, stretching of tendons, voluntary

inversion, supination and dorsal flexion to over-correct the lesion, and later exercises in bare feet to develop the muscles are all of value

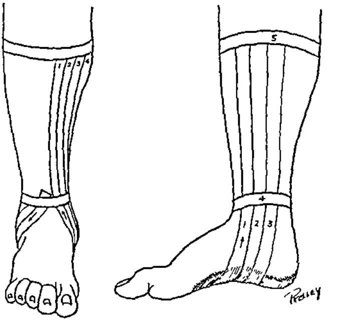


FIG 337 —Strapping of the longitudinal arch useful in acute foot strain Straps 1, 2 and 3 are applied f...

...Lippincott Company)

Treatment of Advanced Flat Foot.—If the arch is flattened only on weight bearing, the same treatment is used, as outlined previously for foot strain, but arch supports are used in all cases temporarily.

In advanced painful flat foot, relief may be obtained by manipulation forcibly to remold the arch of the foot and break up adhesions [9] This type of case should be referred to the orthopedic surgeon who, after breaking up adhesions, will usually apply a cast with the foot in the corrected position [9] After approximately two weeks, removable casts and non-weight-bearing exercises are used until the patient has good control of the arch Arch supports are fitted and weight-bearing exercises are performed In these patients, arch supports are usually worn indefinitely In rigid flat foot due to arthritis, manipulation is sometimes necessary, but not in the acute phase

Surgical procedures may be required.[18] A considerable number of operations have been advised for the treatment of flat foot.[18] Much of the strain in the rigid flat foot takes place at the joint between the astragalus and the os calcis Tendon and ligament operations are inadequate. Operations which stiffen the posterior tarsal joints often combined with a wedge

osteotomy to produce abduction and valgus are the most satisfactory. Such

If patients with advanced flat foot continue to bear weight, an arthritis may develop from constant strain.[56] In young patients with such a

shoe, arch support or braces should be used.

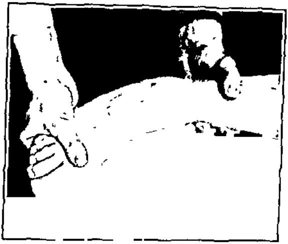


FIG. 338 —Manipulation of the foot. Short leverage is used. The forefoot is inverted, and posterior tarsal joints are manipulated

DEPRESSION OF THE METATARSAL ARCH

fourth is the lowest.

The heads of the first and fifth metatarsals bear weight first followed in order by the fourth, third and second; when weight is relieved, the fourth, third and second metatarsal heads rise to an elevated position.

...mmon in children, but is ...ommonest cause is the

...ults in tenderness and callus under the metatarsal heads, edema of the forefoot, and often leads to cramps in the calves and pain in the anterior part of the lower leg; the depression of these heads removes their shock absorbing action. *Repeated* shocks or trauma to the heads of these bones irritate the soft tissues under them and cause a burning sensation or numbness or tingling in the ball of the foot.

temporary measure; (4) Strapping of the anterior arch or an elastic or leather cuff around the foot behind the metatarsal heads, (5) A metatarsal bar and (6) properly fitting shoes; (7) Exercises to strengthen the flexor

ture of the extensor muscles of the toes A strip of leather approximately ½ inch wide and $\frac{3}{16}$ inch thick running across the outer sole from just proximal to the first metatarsal head to a point proximal to the fifth metatarsal head is attached to the sole or inserted between the layers of the sole.

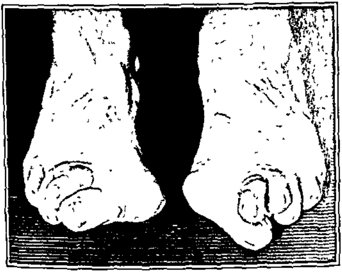


FIG 339 —Hallux valgus, with marked spread of forefoot, exostosis and bursa at first metatarsophalangeal joint

COMMON ABNORMALITIES OF THE FEET

Hammer-toe.[13]—This may begin in childhood and is most common in the second toe (although sometimes it is a congenital defect). It consists of extension at the metatarsophalangeal joint with flexion at the proximal interphalangeal joint; this is usually caused by pressure of a short narrow

early mild cases, and surgical resection in the more advanced cases Exercising the foot, *e g*, flexing the toes over the edge of a book or step, and passively stretching the hammer toe in plantar flexion hastens recovery.

Hallux Valgus.—The great toe is abducted so that it lies on top of, or under the other toes Associated with this is marked prominence of the

y enlargement of the inner side
bursa may form (due to pressure
called a bunion. Inflammation may occur in the bursa, occasionally with suppuration.[52]

Hallux valgus may be present without the formation of a bunion and without marked discomfort even in the presence of marked deformity. Short, narrow, pointed shoes or snugly fitting socks are a common cause of hallux valgus. A congenital metatarsus varus may precede hallux valgus. It is often associated with a depressed metatarsal arch

In mild cases with slight discomfort relief may be obtained by the use of proper shoes, insertion of a pad between the first and second toes, stretching of the great toe, and proper arch supports and exercises. A metatarsal cuff will often correct the alignment of the toe in mild cases; the spreading (splaying) of the anterior metatarsal region must be cared for properly and

of a small portion of the proximal phalanx.[7] There are many surgical procedures for the correction of this deformity.[29]

Hallux Rigidus.—This is a limitation of plantar or dorsiflexion of the great toe usually resulting from bony proliferation at the margins of the metatarsophalangeal joints.[57]

In hallux rigidus, permanent subjective relief may be obtained in early

bent at the tarso-metatarsal junction so that they are turned toward the opposite foot.[34] Although this is often a congenital anomaly, it may be made worse with splaying. These patients experience very little inconvenience if they are supplied with shoes which conform to the shape of their feet (*i. e.*, inflare shoes). With severe deformity, operative correction may be required.

Anterior Metatarsalgia.—This condition may occur in the foot with a high longitudinal arch, the foot with a short Achilles tendon, the abducted foot, or without evident pathologic change; this latter has been called *Morton's metatarsalgia*.[45] It is often seen with a depressed metatarsal arch. It is characterized by the sudden onset (while walking) of a severe crampy pain in the anterior portion of the foot forcing the patient to remove the shoe at once, and to move and rub the toes until the pain is eased.

This is often unilateral, affecting females more frequently than males. It is due mainly to lateral compression of the forefoot forcing the head of the fifth metatarsal inward and behind the head of the fourth, thus causing pressure on the large superficial branch of the external plantar nerve which lies between the heads of these two bones. This results in a neuralgic pain. With prolonged irritation of the nerve a neuroma may develop.[18]

Symptoms of this disorder include: 1. Burning, numbness, tingling or cramplike pain in the forepart of the foot, often under the fourth metatarsal head occurring when wearing shoes (in most instances).

is set up causing pain whether the shoe is worn or not

3 Tenderness and numbness may persist for several days in the forepart of the foot or toes especially about the head of the second, third, or fourth metatarsals

4. Pain may radiate up the foot or leg, or to the end of the toe

5 In some cases, the only discomfort may be an aching across the metatarsal arch.

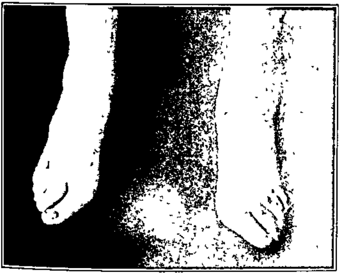


FIG 310 — Hallux valgus with spread of the forefoot, hammer-toe deformity with corns and swelling of the ankles in a patient with rheumatoid arthritis in both feet

6 Painful callus is frequently seen under the metatarsal head

7. Sensory disturbance in the web between the fourth and fifth or third and fourth toes may be found

Treatment consists of the use of felt pads placed just proximal to the head of the fourth metatarsal bone and a metatarsal elastic strap to decrease splaying of the foot, if the pain persists, a metatarsal bar may be tried.

In the treatment of some of these patients it may be necessary to inject from 2 to 3 cc of 1 per cent procaine hydrochloride into the dorsum of the foot or to the painful area near the sole of the foot.

th

fit

the tumor be excised.

Overlapping Toes.[40]—These usually result from narrow shoes (or may be congenital). Such toes may be held in normal position by adhesive strips

If the fifth toe overrides the others markedly a plastic operation may be necessary on the affected toe.

Achillodynia.—This is an inflammation of the bursa which lies between the os calcis and the tendo Achilles at the point of attachment of the tendon.[15] Symptoms include pain over the bursa with swelling, especially during walking. Treatment consists mainly of *rest, removal of pressure* from shoes and heat to the bursa, in some cases it may be necessary to inject this with procaine for relief A felt or sponge rubber pad under the heel affords some relief.

Painful Heel.—This may be caused by traumatic rupture of the Achilles tendon, inflammation of the retrocalcaneal bursa, exostoses, degenerative joint disease, strain on the attachment of the plantar fascia or inflammation of a bursa (occasionally present between the tendo Achilles and the skin) or degeneration of the calcaneal fat pad.

Pain on the sole at the center of the heel on walking may be due to the presence of an exostosis, an inflamed bursa or undue trauma to the tissues in this area.[26] *If no exostosis is present* (as seen by x-ray), *relief is usually obtained by 1 per cent procaine injections (1 to 3 cc) into the painful region and insertion into the heel of the shoe of a soft rubber doughnut, i e*, a piece of rubber approximately ¼ inch thick with an area about 1½ inches in diameter cut out in its center portion. An arch plate may be made with a hole in the central portion of the heel and padding around the edge of the heel.

Painful heels which have developed during the first few weeks of military training are usually relieved by rest and contrast baths and injection of procaine into the painful area. A soft pad or hollowed out heel are helpful measures *Hot foot baths and exercises to obtain good alignment of the* foot should also be given.

Bony Spurs.—*Bony spurs on the anterior plantar surface of the os calcis*[3] *or on the dorsal surface of the foot occur more commonly in the absence of gonorrheal infection than in its presence.* Many result from chronic strain at points of ligamentous attachments. Relief of strain and procaine infiltration usually causes symptoms to disappear When irritation continues they must sometimes be removed. In most instances soft calcareous material will be found at the attachment of the plantar fascia. Bony spurs [illegible]

[illegible]

tendency for the foot to roll outward This may be due to [illegible]
[illegible] ith a short, contracted
[illegible] and especially polio-
[illegible] ermit the development
[illegible] the heel and *ball of the* foot instead of the three usual points, this strains the plantar ligaments and plantar fascia, especially when a low heel is worn.

Symptoms of pes cavus include pain in the *ball of the foot, a sense of* tiredness in the longitudinal arch, cramping in the calf and occasionally backache. *In addition to the* abnormally high longitudinal arch, one notes a tendency of the foot to *roll outward, a shortened* heel tendon, marked prominence of the ball of the foot, contraction of the plantar fascia, tenderness over the heads of the metatarsal bones and over the plantar fascia,

decreased mobility of the foot and the formation of callus over the metatarsal heads[17]

enough forward to elevate the distal portion of the metatarsal bones and thus relieve pressure on the metatarsal heads. Exercises and physical therapy are of value. In the severe cases a wedge osteotomy on the dorsum of the foot and transplantation of the extensor tendons to the metatarsal bones may be necessary[18]

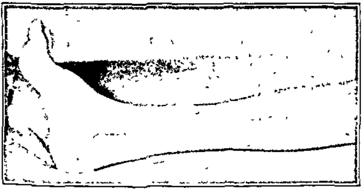


FIG 341 —Cavus of the foot There is contracture of the plantar fascia The dorsal portion of the mid-foot is prominent The toes are held in extension

IMMERSION FOOT—TRENCH FOOT

Immersion foot (and its counterpart on land—trench foot) are due to prolonged exposure of the dependent lower extremities to cold and moisture[40]

Individuals with immersion foot show the member to be swollen, blue in color and blistered Recovery is usually complete even though massive gangrene from the ankle down may appear imminent. With recovery there is usually an early hyperemic stage and a later period of fibrosis With severe and prolonged exposure, pain may be severe in both of these stages and may cause prolonged incapacity.

As soon as the legs become warm and the circulation recovers, the hyper-

tissues as evidenced by petechial hemorrhages due to ruptured capillaries and marked congestion of the feet when dependent. When the nerves recover, a burning pain develops. The toes may remain anesthetic for prolonged periods although recovery of sensation may take place rapidly in the proximal parts of the feet

The circulation returns to normal and the hyperemia subsides in from one to four weeks. The blebs absorb and the skin peels off with some scarring.

Persistent disability may be due to the fibrosis which follows chilling short of frostbite. The amount of scar tissue reaction depends upon the initial exudation of fibrin and tissue anoxia.

Malnutrition and avitaminosis may play a rôle in the pathogenesis of these painful, swollen feet.

Men with immersion foot must be kept off their feet. The skin should be protected against rupture of the blebs and pressure sore formation in the weight-bearing portions of the anesthetic skin. If streptococcal infection [illegible] ower legs, moist

[illegible] uld be warmed. A high protein, high vitamin, nutritious, easily digested diet should be prescribed as soon as tolerated. Treatment for shock may be necessary in some individuals.

The injured feet must be kept cool (while the body is being warmed). Cooling of the feet can be accomplished by exposing the legs below the knees to a cool room temperature, and by allowing cool air from an electric fan or blower to pass over them. *Do not rub these feet or paint with a strong disinfectant!* These regions should be treated as a second degree burn with aseptic precautions, even including antibiotic therapy, a booster dose of tetanus toxoid and masking of the dresser's nose and mouth. This treatment is continued (for a week or more) until the hyperemia has subsided and circulation through the thrombosed subcutaneous vascular bed has recovered. The ideal cutaneous temperature is between 70° and 75°, at which point pain is relieved. Such cooling lowers the metabolic need for oxygen sufficiently so that adequate quantities can be supplied through the injured capillary bed With cooling of the feet, there is improvement in the appearance of the skin, shrinking of the blisters and relief of pain.

In severe cases, sterile pledgets of cotton are placed between the toes, the foot is covered with a sterile towel and thoroughly dried ice bags are then placed around each foot and over the towel Treatment may be required for several days or a week or two; with subsidence of the hyperemia, less ice is required and cool air alone may be adequate.

The skin desquamates as the circulation returns to normal and blebs absorb. Disability may last for months in severe cases although mild cases [illegible] is due to

[illegible]

result in some improvement, in some cases no treatment is particularly [illegible] tissues

[illegible]

Immersion foot has a [illegible]
have cold clammy feet.
chronic foot strain or an [illegible]
condition produces no s[illegible]

common In cold weather, however, the skin on such feet does not withstand freezing weather well unless wool socks and good shoes are worn Serious lesions are rare but painful superficial necrosis is fairly common This is commonly called chillblain or frostbite. The early treatment is milder but similar to that for immersion foot The venous congestion should be corrected

INFLAMMATORY LESIONS OF THE FOOT

The common inflammatory disorders of the skin of the feet are contact dermatitis, pyoderma, paronychia, psoriasis, lichen simplex chronicus, disturbances of the sweat glands, persistent erythema of the soles, radiodermatitis, urticaria, granuloma annulare, acrodermatitis perstans,

foot, sporotrichosis, blastomycosis and chromoblastomycosis)

The great number of drugs recommended indicates that there is no specific cure for dermatophytosis. In mild intertriginous infection, 2 per cent salicylic acid in borated talc may be effective In acute vesicular cases, relief may be obtained by removing the tops of vesicles with scissors and applying cold wet dressings of 1 to 2000 aqueous solution of potassium permanganate. In severe infections, the patient should stay off of this feet With subsidence of the acute process, potassium permanganate foot baths 1 to 2000 may be continued and a diluted ointment of benzoic and salicylic acid may be applied, as well as fungicidal agents, such as Castellani's paint or 2 per cent gentian violet. In chronic infections, roentgen therapy may be of value. Infected toe-
Whitfield's ointment and
nails before applying me
cautiously over the nails
inasmuch as the new nails tend to be infected

Blastomycosis occasionally involves the foot. The lesion usually begins as a small papulopustule which becomes crusted and gradually enlarges by peripheral extension producing sharply outlined patches limited by a rounded

of the lesion will show the fungi under the microscope. Small lesions may be excised ssium iodide by mouth and

gous infection involving the upper layers showing a predilection for the foot. It is rare in the United States. In most instances the disease has occurred in men who walk barefooted on a farm. The incubation period is very variable, from a month to several years.

The diagnosis can usually be made early in the disease by the presence of discharging granules and by the demonstration of the fungus in these granules (microscopically). Although large doses of potassium iodide may be helpful in the early stages of the process, cure has been obtained in most of the reported cases only by amputating the infected foot.

Ainhum is a syndrome characterized by a constriction at the base of a toe, usually the fourth or fifth toe. A deep groove is formed in the plantar fold. The etiology of the condition is unknown and the disease usually progresses to ulceration or gangrene. Spontaneous amputation may occur; in other cases, surgical amputation of the affected toe may be done.

Tumors of the foot are frequently multiple. These include foreign body granuloma; pyogenic granuloma; angioma; angioblastoma; angioneuromyoma; Kaposi's sarcoma (idiopathic multiple hemorrhagic sarcoma); *synovial cysts, ganglion; fibroma; xanthoma, melanoma; carcinoma;* and sarcoma (which is relatively uncommon on the foot).

CONGENITAL DEFORMITIES ABOUT THE FOOT

Of these there are many that are rare. The common ones are club foot, deformities of the toes, *supernumerary bones and congenital relaxation of* the foot.[25]

Club foot, sometimes called talipes equino varus is a deformity of the foot present at birth showing varus equinus and forefoot adduction. It becomes progressively worse if untreated and eventually makes walking almost impossible. Early and persistent orthopedic treatment is required.

The congenital deformities of the toes require the same type of treatment *as those which are not congenital. Supernumerary bones may occur about* all of the tarsal bones. Thirty-nine different types have been described These are sometimes confused with fractures. The commonest one is an accessory scaphoid bone. At times the tendon of the tibialis anterior muscle attaches to this bone. This weakens the pull of the invertor muscles and aids pronation of the foot. Usually exercises, proper shoes and arch supports relieve symptoms when they are present.

Congenital relaxation of the foot is sometimes called congenital flat foot Many small children have weakness and other disturbances of the feet[16] Many of these disappear spontaneously when the child develops better balance and skill in walking. A few show persistent ligamentous relaxation of the feet with displacement of the medial tarsal bones downward and inward on weight bearing. In some instances the dorsum of the foot can be brought to the anterior surface of the tibia. This disability persists and becomes worse if untreated. *Treatment consists of night boots* holding the feet in varus and equinus. These may have to be worn for six months [illegible] for the depressed arches and a raise

arely needed and are not undertaken before the age of nine

CIRCULATORY DISTURBANCES IN THE FEET

These may be of two kinds, venous and arterial.[19] Disturbances in the

baths are of value. A pressure bandage or an elastic stocking may be necessary to aid the superficial circulation. Operations which obliterate the most tortuous of the superficial veins are sometimes indicated. *Inflammation in the veins, called phlebitis*, may cause pain particularly on the dorsum of the foot and about the malleoli. There is local tenderness, heat and redness of the skin. There is always inflammation in more proximal veins. If pain is severe rest and elevation of the foot should be prescribed. Pres- [illegible]

of vasospasm is that seen in Raynaud's disease, although a number of traumatic disturbances and occasionally psychosomatic disturbances can



FIG 312.—*Leather cuff worn about the forefoot* for spreading of the anterior arch and flexion deformity of the toes (Kuhns, courtesy of Rheumatism.)

produce this picture. The milder ones often require no treatment. The more severe ones sometimes require sympathetic block or sympathectomy.

When the arterial channel becomes narrowed by degenerative processes one of the earliest symptoms of an impaired blood supply is a cramp-like pain in the muscles of the leg on walking. This is called *intermittent claudication*. Later coldness, sensory changes and superficial gangrene may appear. Exercises, both postural exercises and foot exercises, are of value. Well-fitting shoes and stockings are essential. Any break in the skin should receive immediate attention. The skin should be kept soft by lanolin or olive oil as required. Occasionally, lumbar sympathectomy improves circulation somewhat, but usually the individual must lessen his activities to conform to the decreased arterial supply.

us
Tl
to [illegible] of gangrene. Spontaneous amputation may occur; in other cases, surgical amputation of the affected toe may be done.

Tumors of the foot are frequently multiple These include foreign body granuloma; pyogenic granuloma; angioma, angioblastoma, angioneuromyoma; Kaposi's sarcoma (idiopathic multiple hemorrhagic sarcoma), synovial cysts, ganglion; fibroma; xanthoma; melanoma; carcinoma; and sarcoma (which is relatively uncommon on the foot).

CONGENITAL DEFORMITIES ABOUT THE FOOT

Of these there are many that are rare. The common ones are club foot, deformities of the toes, supernumerary bones and congenital relaxation of the foot [25]

Club foot, sometimes called talipes equino varus is a deformity of the foot present at birth showing varus equinus and forefoot adduction. It becomes progressively worse if untreated and eventually makes walking almost impossible. Early and persistent orthopedic treatment is required.

The congenital deformities of the toes require the same type of treatment as those which are not congenital. Supernumerary bones may occur about all of the tarsal bones. Thirty-nine different types have been described. These are sometimes confused with fractures. The commonest one is an accessory scaphoid bone At times the tendon of the tibialis anterior muscle attaches to this bone This weakens the pull of the invertor muscles and aids pronation of the foot Usually exercises, proper shoes and arch supports relieve symptoms when they are present.

Congenital relaxation of the foot is sometimes called congenital flat foot. Many small children have weakness and other disturbances of the feet.[46] Many of these disappear spontaneously when the child develops better balance and skill in walking. A few show persistent ligamentous relaxation of the feet with displacement of the medial tarsal bones downward and inward on weight bearing. In some instances the dorsum of the foot can [illegible]

When the child begins to walk, [illegible] [illegible]hes and a raise [illegible] Exercises are [illegible] good habitual position of the feet in standing and walking. Operative procedures are rarely needed and are not undertaken before the age of nine

CIRCULATORY DISTURBANCES IN THE FEET

These may be of two kinds, venous and arterial.[19] Disturbances in the venous circulation are often found in older persons associated with varicose veins. Unless ulceration occurs, these rarely cause pain in the foot; but there may be swelling and a sense of heaviness. Exercises and contrast

baths are of value A pressure bandage or an elastic stocking may be necessary to aid the superficial circulation. Operations which obliterate the most tortuous of the superficial veins are sometimes indicated. *Inflammation in the veins, called phlebitis,* may cause pain particularly on the dorsum of the foot and about the malleoli There is local tenderness, heat and redness of the skin. There is always inflammation in more proximal veins. If pain is severe rest and elevation of the foot should be prescribed Pres-



Fig 342 —*Leather cuff worn about the forefoot* for spreading of the [illegible] flexion deformity of the toes (Kuhns, courtesy of Rheuma[illegible]

produce this picture The milder ones often require no tr[illegible]

[illegible]

on

pa

claudication Later coldness, sensory changes and sup[illegible] appear. Exercises, both postural exercises and foot [illegible] Well-fitting shoes and stockings are essential. Any [illegible] receive immediate attention The skin should be [illegible] olive oil as required Occasionally, lumbar [illegible] circulation somewhat, but usually the individual to conform to the decreased arterial supply.

ARTHRITIS INVOLVING THE FEET

The joints of the hands and f...

t

...will often prevent practically all disability of the feet in arthritis ...

... be due to: localized pressure, strain ... chronic local inflammatory changes ... or knee, or faulty posture upon the function of the foot must be considered Actual arthritis of the small joints of the feet may produce local pain Stiffness of various joints may result with atrophy of the small muscles of the foot and eventual production of claw-shaped toes, dislocations, corns and calluses.[9]



FIG 343



FIG 344

FIG 343 —Ankle strap to support the midfoot and relieve strain about the ankle (Kuhns, courtesy of Rheumatism)

FIG 344 —Cuff of plaster which can be worn about the forepart of the foot to prevent spread of the forefoot and to aid flexion of the toes (Kuhns, courtesy of Rheumatism)

... is a ...ted

ing over the affected joints and pain on motion. Later, muscular spasm leads to stiffness and deformity about the toes and tarsal joints In the terminal stages, adhesions, ankylosis and bony deformity may occur.

... of the forefoot ...algus posi-
...trophy of
...mal inter-

phalangeal joints, extension at the metacarpophalangeal joints, lateral

deviation of the proximal phalanges and hallux valgus (For treatment see Chapters on Prevention and Correction of Arthritic Deformities)

OPERATIVE TREATMENT

This is not often required in the arthritic foot unless treatment has been neglected.
the arthritis
followed by
arthritic deformity Manipulation of the foot to rupture adhesions may be necessary, this is performed best under general or spinal anesthesia to permit muscular relaxation.[46] In manipulation of the foot the deformed portion of the foot is brought further into deformity and then is slowly
ulations do
be necess-
attempt is
made to get the foot into a normal weight bearing position.[63]

After-care of Manipulated Feet —Maintain the foot in a corrected position by adhesive strapping or plaster cast and apply heat to lessen pain and edema Elevation of the foot is sometimes helpful With subsidence of the reaction, use massage and exercises, arch support (if required) and proper shoes. If extensive adhesions have been torn, the patient should remain in bed for several days

OTHER ABNORMALITIES OF THE FOOT

Plantar Fascitis.—Inflammation of the plantar fascia may cause pain in the feet. In about one-half of these patients, there is a preceding influenza or sore throat; in other cases, a focus of infection has been suspected Metabolic disturbances and endocrine dysfunction particularly at the menopause may be found with this condition. Overactivity (excessive walking) may lead to this condition. It produces tenderness of the entire plantar fascia coming on several weeks after an acute infection The pain in later stages is localized to the region of the heel Swelling may also be noted.

In those cases following an acute infection, spontaneous cure usually results within a month or two. In other instances, the process may become chronic and persist for years

Treatment of plantar fascitis includes *radiant heat locally*, use of a *rubber pad to protect the heel, procaine injections into the plantar fascia* for temporary relief of discomfort and *proper support* to the foot with *exercises* later to strengthen the foot.

March Foot.—March foot (also called march fracture) is found in one or more metatarsal bones most often in young adults probably as the result of prolonged foot strain[20]

m

we

an

walking on such a foot may result in an impacted or complete fracture with

displacement of the fragments Fatigue with gradual disruption of *osseous tissues is probably the chief factor in its causation.*

Roentgen examination in the early stage discloses slight fuzziness due to the formation of subperiosteal new bone on one aspect of the neck of these bones; later, definite new bone may be seen by roengen examination and the mass may be felt. In some of these patients, a fracture may be found at this site. All of these heal *within one to two months. There* is no specific therapy.[36]

In patients with complete or displaced fractures, a plaster cast is applied for approximately three weeks; the individual is disabled for three to four weeks. Walking casts may prove useful. An anterior arch pad with

epiphyseal changes *in the bones of the foot seen by roentgen-ray include:*[7] tarsal scaphoiditis[24] (Kohler); involvement of the epiphysis of the os calcis (apophysitis), infraction of metatarsal heads (Freiberg)[20] It has been noted that all of these follow trauma, usually occur in growing children, involve epiphyses which are ununited, and are frequently accompanied by rarefaction, fragmentation or abnormal calcification in epiphyses.

Epiphysitis of the Heel (Calcaneal Epiphysitis).[18]—The epiphysis of the heel is subject to direct trauma and strong lateral pull from muscles.

Inflammation occurs most frequently in boys ten to seventeen years of age. Involvement is usually bilateral; history of trauma may or may not be found.

Symptoms include pain at the back of the heel, aching along the *Achilles* tendon or calf muscles and pain on climbing stairs.

Examination shows a tendency to walk upon the toes to relax the pull of the Achilles tendon, with marked localized tenderness over the attachment of the tendo Achillis, tenderness of the heels posteriorly, and limitation of *dorsiflexion with pain on forced motion.*

Roentgen findings include: a fluffy, moth-eaten, flattened or fragmented epiphysis of the heel seen in the lateral view; marked irregularity of the adjacent *posterior surface of the calcaneum with a punched-out appearance;* and a cloudy irregular epiphyseal line.

Symptoms *disappear* when the *epiphysis* unites with the calcaneus (at about seventeen years); the epiphysis of the heel appears at about the age of ten.

heels by a pad of felt or the tendo Achillis; firm t to the feet may relieve on is permitted until the sional resistant case, the foot must be immobilized in slight plantar flexion for several weeks with plaster of Paris casts

Kohler's Disease.—This is an uncommon juvenile type of osteochondritis, beginning insidiously most often in males from two and one-half to seven and one-half years of age. The essential *pathologic finding is a* distortion of the nucleus of ossificati e tarsal scaphoid. Symptoms include localized pain, swelling a ss on the dorsum of the foot (over the affected scaphoid) and

Roentgen examination reveals marked flattening and irregular outline of the calcified area of the scaphoid, narrowing in the anteroposterior diameter and increased density as though it had been crushed Roentgen appearances become normal eventually The condition is most frequently unilateral A normal foot is usually obtained as complete regeneration of the involved bone occurs in an average of less than three years

No treatment is required except protection against excessive trauma as *normal bone formation always occurs; symptoms may be relieved by local* strapping or arch support.

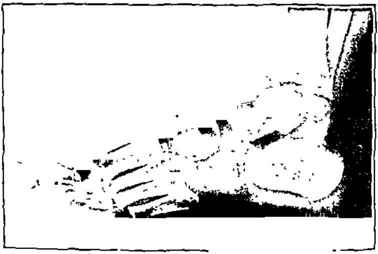


FIG. 315.—Kohler's Disease Note thin, dense tarsal scaphoid

Freiberg's Disease.—This is a juvenile form of osteochondritis usually affecting the head of the second metatarsal bone. Symptoms include pain, tenderness, and later, bony swelling in the forefoot Typical roentgen findings are flattening of the head of the second metatarsal bone, followed by areas of sclerosis in the subchondral bone and expansion of the head by osteophytes No treatment is required unless there is pain or limp on walking. Conservative therapy includes the use of an anterior arch pad or a thick metatarsal bar on the shoe to prevent weight bearing on the affected area.

BIBLIOGRAPHY

1 ADAMS, J D · Jour Am Med Assn, **92**, 1773, 1929
2 AITKEN, J T · Brit Jour Phys Med, **12**, 92, 1949
3 ARMSTRONG, G A Canadian Med Assn Jour, **42**, 226, 1940
4 BAILEY F T [illegible] 1946
5 BAKE [illegible]
6 BELL [illegible]
7. BINGC [illegible] nd Joint Surg, **32**, 214, 1950
8 BIVINGS, L. Am Jour. Dis Child, **76**, 440, 1948.
9 BRETT, A L Physiotherapy Rev, **21**, 306, 1941
10 BROWN, J E: Arch Phys Med, **31**, 101, 1950.
11. BRUCE, J, and WALMSLEY, R Lancet, **2**, 656, 1938

12 CARTER, H Med. Officer, 80, 147, 1948
13 CLEVELAND, M, and WINANT, E. M.: Jour Bone and Joint Surg, 32, 163, 1950
14 COMPERE, E L. Med Clin N A, 30, 635, 1946
15 COVENTRY, M B Surg Clin N. A., 28, 1079, 1948
16 CREER, W S: Brit Jour Industrial Med, 1, 54, 1944
17 CUTTLE, T D. U S Naval Med Bull, 41, 216, 1943
18. DICKSON, F D, and DIVELEY, R L Functional Disorders of the Foot, Lippincott, Philadelphia, 1949
19 FETT, H. C, and POOL, C C Am Jour Surg, 78, 522, 1949
20 FREIBERG, A H Jour Bone and Joint Surg, 8, 251, 1926
21 GALLAGHER, J R, and DELORME, T L Jour. Bone and Joint Surg, 31, 847, 1949
22 GIKLING, G Am Jour Nursing, 49, 786, 1949
23 GOOD, M G.: Practitioner, 163, 229, 1949
24 GRAHAM, J Illinois Med Jour., 75, 425, 1939
25 GUNTZ Ztschr f. Orthop, 77, 154, 1947.
26 HARRIS, R I, and BEATH, T.. Jour Bone and Joint Surg, 31, 553, 1949
27 HARTLEY, J *Mil Surgeon, 96, 177, 1945.*
28 HAUSER, E D W D Indust Med, 18, 326, 1949
29 JOPLIN, R J Jour. Bone and Joint Surg, 32, 779, 1950
30 KARK, W Jour Royal Army Med Corps, 77, 166, 1944
31 KEY, J A: Am Jour Surg, 79, 667, 1950
32. KING, L S Am *Jour Clin Path, 16, 124, 1946.*
33 [illegible]
34
35 [illegible] s, 10, 252, 1940
36
37
38
39
40 [illegible] 47
41
42
43.
44
45.
46 [illegible] urchill, London, 1949
47 MEYER, O Med. Record, 157, 732, 1944
48 [illegible] Am Med Assn 109 1112 1937
49 [illegible] 7, 1942.
50
51
52
53 [illegible] 9, 431, 1947.
[illegible]
[illegible] ce and Obst, 66, 663, 1938
[illegible] 45
58 [illegible]
59 WATSON-JONES, R Jour Bone and Joint Surg, 31, 650, 1949
60 [illegible] New England Jour Med, 232, 415, 1945
61. [illegible] 47, 1940
62.
63

CHAPTER 57

LOW BACK PAIN AND SCIATICA

By Philip D. Wilson, M.D. and Philip D. Wilson, Jr., M.D.

INTRODUCTION

logic confirmation Twenty or thirty years ago the answer to mechanical low back pain was the "slipped sacroiliac" Now, the fad is for the "slipped disc"

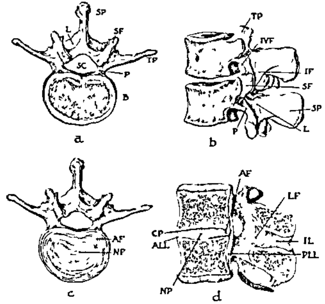


Fig 316—Anatomy of the lumbar vertebræ and their articulations

A Superior view of a stripped lumbar vertebra B Lateral view of two articulated lumbar vertebræ C Superior view of a horizontal section of a lumbar disc D Lateral view of a sagittal section of two articulated lumbar vertebræ

Key B—body
SC—spinal canal
IVF—intervertebral foramen
IF—inferior articular facet
SF—superior articular facet
P—pedicle
TP—transverse process
SP—spinous process
L—lamina
AF—annulus fibrosus
NP—nucleus pulposus
CP—cartilage plate
ALL—anterior longitudinal ligament
PLL—posterior longitudinal ligament
LF—ligamentum flavum
IL—interspinous ligament

intraperitoneal and retroperitoneal structures must not be forgotten, and the internist will often play an important part in diagnosis and selection of treatment. There is no field in which the ingenuity of the attending physician is taxed to a greater degree.

I. ANATOMY

The structural details of the back are exceedingly intricate, but it is impossible to understand, diagnose and treat lesions of the spine without a rather fundamental knowledge of its parts

Anatomical Considerations.—Each vertebra is composed of an anterior portion, the body, and a posterior part, the neural arch From the latter there are several processes of importance. These are the transverse processes, one on each side, superior and inferior articular processes (facets), one above and below on each side; and one spinous process. The portion of the neural arch connected to the body and anterior to the articular processes is called the root or pedicle, that behind is called the lamina; that part between the superior and inferior articular facets has come to be known as the pars interarticularis or isthmus.

The vertebræ are articulated together by a system of joints three for each level. Between the vertebræ there is a synchondrosis commonly known as the disc The disc is composed of a tough fibrocartilaginous outer

annulus and from annulus to nucleus are gradual Posteriorly, one for each side, there are true diathrodial joints. These lie between the inferior facets of one vertebra above and the superior facets of the next below.

The ligamentous support of the spine is provided by the anterior longitudinal and posterior longitudinal ligaments Of these the anterior is the stronger and the posterior fails to meet it laterally in the region of the intravertebral foramen In addition there are the ligamentum flavum, an elastic structure which bridges the interlaminal spaces and reinforces the facet joint capsules, and the interspinous, intertransverse and iliolumbar ligaments.

From an embryological point of view the anlage of the spine forms in a right and left column on each side of the primitive notochord just anterior to the neural tube.[3] At a later stage of development the two halves fuse anteriorly and posteriorly around the developing spinal cord. It is failure of this process that accounts for the relatively frequent incidence of fusion defects, various degrees of spina bifida when posteriorly, and various degrees of platyspondyly when anteriorly The latter condition has a much rarer incidence than the former.

The bony structure of the vertebra is formed also from a fusion of two parts in another plane, a process that proceeds simultaneously with the former; that is the lower portion of one sclerotome with the subjacent

one part of another. Disturbances of this stage of de-

chord is figuratively squeezed out and forced above and below where it concentrates to aid in the formation of the nucleus pulposus This process

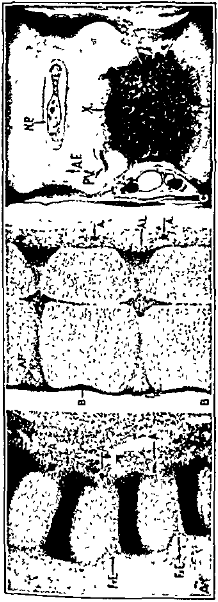


Fig 347 —Development of the vertebral column

A, Four week (1 1 cm) embryo Lateral view of a sagittal section The dense portion of the sclerotome is in the cranial half and the less dense portion in the caudal half of the developing vertebra The disc will develop at the level marked FVE (Fissure von Ebner) The fissure is dividing the future vertebral bodies The inter-egmental arteries (A) are also seen

B Nine week (4 cm) embryo Displacement of notochordal cells (N) into the disc zones (D) The anterior longitudinal (AL) and posterior longitudinal ligaments are differentiating The anlage of the annulus fibrosus (AF) is also seen. The vertebral bodies (B) are made of cartilage at this stage of development

C, Seven and one-half month (27 cm sitting height) fetus well formed intervertebral disk and vertebral body Annulus fibrosus (AF) well developed Notochordal cells forming nucleus pulposus (NP) The cartilagenous anlage of the vertebra is undergoing endochondral ossification (X and Y) (Ehrenhaft, courtesy of Surgery, Gynecology and Obstetrics)

is often only accomplished imperfectly leaving defects in the cartilaginous plates. The annulus fibrosus which encloses the nucleus pulposus has direct origin from the mesenchyme left as the cephalad and caudad portions of the sclerotome separate to take part in the formation of the bony vertebræ.

The anatomical details of the sacrum do not warrant special description. The articulation between it and the fifth lumbar vertebra follow the pattern described above. The sacrum articulates with the pelvis by means of the two sacroiliac joints which are partly fibrous and partly diarthrodial The surfaces of the fibrous portion of these joints are irregular and fit closely and snugly together.

In the female pelvis there is less bony apposition than in the male and the ligaments are stronger. In both sexes some motion can and does take place, but the range is small and physiologically insignificant The motion is in a rotary plane and is resisted in one direction by the wedge shape of the sacrum and in the other by the check of the sacrotuberous and sacrospinous ligaments. The cartilaginous surface of the sacrum is thicker and hyaline, that of the ilium thinner and fibrous. The joint is supported by some of the densest ligaments of the body. For this reason it is doubtful that sacro-iliac strain or "slipping" can occur, without extensive injury (i e., fracture of pelvis) or disease. However, that portion of the joint which is lined, although imperfectly, by synovium is subject to all the diseases of diarthrodial joints.

The coccyx is composed of four segments, the first one of which is often free. Motion can and does take place between the sacrum and coccyx There is little ligamentous support of this joint and it is vulnerable to injury, but it is so situated as to be fairly well protected.

The neural arch of each vertebra encloses the spinal cord, together with its supporting structures Originally, the distal end of the cord extends to the caudal end of the dural sac at about the level of the third sacral segment, but it retracts upward as the spine grows in length to reach a final adult position at the level of the first lumbar vertebra. The nerve roots of the lumbosacral plexus are drawn up with the cord and lie within the dura forming the cauda equina. A slender filament, the filum terminale, loosely anchors the distal end of the cord to the coccyx. Cephalad the cord is held by its origin from the brain. It is also fixed by the succession of spinal nerves which pass out through the intervertebral foramina It is supported everywhere by its dural and arachnoidal membranes. Over this relatively immobile neural tube the spinal segments must move smoothly to perform their physiological functions.

Each nerve root leaves the spinal canal under its corresponding vertebral arch except in the cervical spine, where it leaves above That is to say, the first cervical nerve root passes out *above* the first cervical vertebral arch whereas the fifth lumbar nerve root leaves the canal *below* the fifth lumbar vertebral arch. The difference is made up by the presence of eight cervical nerves as opposed to seven cervical vertebræ, the 8th cervical nerve passing out the intervertebral foramen between C7 and T1.

A nerve root is in intimate relation with several parts of the vertebra and discs as it passes from the dural sac to without the neural canal.[8 11 20] In the lower lumbar spine each nerve root together with a sheath of dura becomes separated from the remainder of the cauda equina at the level of

the disc above, courses laterally as it passes distally across its corresponding vertebral body and reaches the intervertebral foramen as it lies opposite its disc of the same numerical designation For instance, the fifth lumbar nerve leaves the cauda equina just above the level of the fourth lumbar disc (the disc between L4 and L5), passes posteriorly to the 5th lumbar body, coursing laterally to the intervertebral foramen between L5 and the sacrum. In the foramen it is held against the medial and inferior surfaces of the pedicle (in this case, of L5) lying in a groove called the sulcus nervi

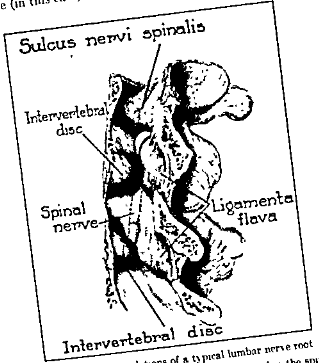


FIG 348 —The relations of a typical lumbar nerve root

View of the 3rd and 4th intervertebral foramina from within the spinal canal The vertebræ have been cut tangentially Note the relation of the 4th lumbar nerve root (dotted lines) to the 3rd lumbar disc At this level the root has less space than it does when opposite the 4th lumbar disc It is thus more readily affected by protrusion of the 3rd lumbar disc than one of the 4th Note also its relation to the facets, here hidden by the underlying portion of the capsule, the fibers of which blend with those of the ligamentum flavum The nerve root is also in close contact with the pedicle (Naffziger, Inman and Saunders, courtesy of Surgery, Gynecology and Obstetrics)

spinalis Anteriorly, lies the body (of L5), inferiorly the bulging (5th lumbar disc and posteriorly shielded by ligamentum flavum, the inferio facet (of L5) Although, the nerve root moves fairly freely in the inte vertebral foramen, pathologic or traumatic disturbances of any of the structures (pedicle, body or facet) can obstruct its passage and ca pressure. It is important to remember that in the lumbar spine the are related to two discs, the one above and the one below i vertebra.

is often only accomplished imperfectly leaving defects in the cartilaginous plates. The annulus fibrosus which encloses the nucleus pulposus has direct origin from the mesenchyme left as the cephalad and caudad portions of the sclerotome separate to take part in the formation of the bony vertebræ.

The anatomical details of the sacrum do not warrant special description The articulation between it and the fifth lumbar vertebra follow the pattern described above. The sacrum articulates with the pelvis by means of the two sacroiliac joints which are partly fibrous and partly diarthrodial. The surfaces of the fibrous portion of these joints are irregular and fit closely and snugly together.

In the female pelvis there is less bony apposition than in the male and

the sacrum and in the other by the check of the sacrotuberous and sacrospinous ligaments. The cartilaginous surface of the sacrum is thicker and hyaline, that of the ilium thinner and fibrous. The joint is supported by some of the densest ligaments of the body. For this reason it is doubtful that sacro-iliac strain or "slipping" can occur, without extensive injury (*i e.*, fracture of pelvis) or disease. However, that portion of the joint which is lined, although imperfectly, by synovium is subject to all the diseases of diarthrodial joints.

The coccyx is composed of four segments, the first one of which is often free Motion can and does take place between the sacrum and coccyx. There is little ligamentous support of this joint and it is vulnerable to injury, but it is so situated as to be fairly well protected.

The neural arch

its supporting stru

the caudal end of

ment, but it retracts upward as the spine grows in length to reach a final adult position at the level of the first lumbar vertebra. The nerve roots of the lumbosacral plexus are drawn up with the cord and lie within the dura forming the cauda equina. A slender filament, the filum terminale, loosely anchors the distal end of the cord to the coccyx Cephalad the cord is held by its origin from the brain. It is also fixed by the succession of spinal nerves which pass out through the intervertebral foramina. It is supported everywhere by its dural and arachnoidal membranes Over this relatively immobile neural tube the spinal segments must move smoothly to perform their physiological functions

Each nerve root leaves the spinal canal under its corresponding vertebral arch except in the cervical spine, where it leaves above. That is to say, the first cervical nerve root passes out *above* the first cervical vertebral arch whereas the fifth lumbar nerve root leaves the canal *below* the fifth lumbar vertebral arch. The difference is made up by the presence of eight cervical nerves as opposed to seven cervical vertebræ, the 8th cervical

tebra

6 19 20

In the lower lumbar spine each nerve root together with a sheath of dura becomes separated from the remainder of the cauda equina at the level of

the disc above, courses laterally as it passes distally across its corresponding vertebral body and reaches the intervertebral foramen as it lies opposite its disc of the same numerical designation. For instance, the fifth lumbar nerve leaves the cauda equina just above the level of the fourth lumbar disc (the disc between L4 and L5), passes posteriorly to the 5th lumbar body, coursing laterally to the intervertebral foramen between L5 and the sacrum. In the foramen it is held against the medial and inferior surfaces of the pedicle (in this case, of L5) lying in a groove called the sulcus nervi

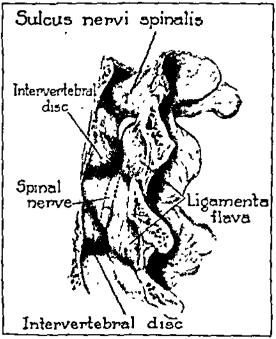


FIG 348 —The relations of a typical lumbar nerve root

ver
(d
wh
the
by
lig
Int

spinalis Anteriorly, lies the body (of L5), inferiorly the bulging (5th) lumbar disc and posteriorly shielded by ligamentum fl...
facet (of L5)
vertebral foram
structures (ped ... and cause
pressure It is important to remember that in the lumbar spine, the nerves are related to two discs, the one above and the one below its corresponding vertebra.

With this preliminary sketch of the individual parts of the spine it now remains to join the whole together as a functional unit.

Spinal Function.—*In utero the spine is formed* in one long curve with its convexity posteriorly. After birth and with the gradual assumption of vertical posture, this curve is altered. Because the thoracic segment is relatively immobile, due to its attached ribs, the kyphosis (curve with convexity posteriorly) persists in this area. However, as strength develops to allow the infant to raise its head and then sit and finally stand, lordoses (curves with convexity anteriorly) develop first in the cervical segment and then in the lumbar segment. By this means balance is achieved and the weight of the trunk is carried directly over its base. As

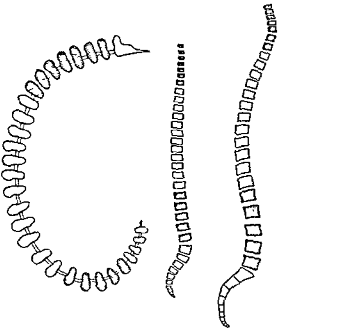


FIG 349.—Spinal posture at various ages. *a*, Six week embryo *b*, Newborn *c*, Adult Note the development of the cervical and lumbar lordotic curves (blackened segments) (Arey, Developmental Anatomy, courtesy of W. B Saunders Company.)

long as the curves anteriorly and posteriorly balance each other, the erect position can be maintained with surprisingly little muscular effort. The lordotic curves are entirely functional, and can be reversed by bending forward, in contrast to the kyphotic thoracic curve which is relatively fixed.

There are two essential forces which are responsible for the development of these curves One is the downward pull of gravity and the other the extending force of the massive erector spinæ However, once the spine is balanced in the erect position, the muscular forces in various directions become purely stabilizing in effect. Thus, if the normal erect posture is not disturbed, it is possible to stand without muscular effort and the ligaments act mainly as check reins to prevent excessive motion.

In the cervical region, this motion is remarkably free in all planes. The vertebral bodies are small, the discs relatively thick and the planes of the

diameter, but the facets are oriented to guide motion mainly in the direction of rotation and lateral inclination. In the lumbar spine motion is largely in flexion and extension. On bending forward, the lumbar lordosis is reversed and on extension it is accentuated. Lateral inclination is only

the muscle groups on one side result in lateral inclination. Rotation is produced by more complex muscle combinations. It is important to remember, however, that since many of the spinal motions are performed in the erect position, that the force of gravity can never be discounted and often the muscles opposing the motion performed are the ones called into play in order to prevent loss of balance. For instance on bending

in order to

presents a

a basis for

The Nature of Pain in Somatic Structures.—There is one more basic item to be discussed in relation to low back pain in order to understand it more clearly and that is the source and character of the pain itself.[12, 13, 14, 16, 17]

The foremost differentiation to be made is that between pain from deep skeletal structures and pain from direct nerve pressure. Almost everyone is familiar with the nature of the latter as seen in the "funny or crazy bone" sensation in which the ulnar nerve is suddenly bumped. Faradic stimulation of a peripheral mixed nerve results in the same sensation. The onset of pain is rapid, it follows the distribution of the sensory portion of the ulnar nerve and is associated with numbness and tingling when severe. If we substitute for the peripheral mixed ulnar nerve, a single spinal nerve root, we will find that the sensation is the same in character, but confined to the peripheral representation or dermatome of that nerve root. The pain is sharp, lancinating and accompanied by paresthesia, and numbness. It may be accompanied by muscle cramps.

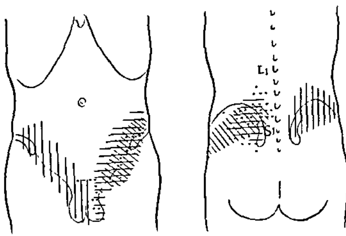
If the pressure is severe and prolonged the nerve is deadened, the property of conduction is lost even though the fibers are still in continuity. The larger nerve fibers, that is, those mediating touch and motor stimuli, lose their function, first followed in order by the progressively smaller ones. Pain fibers, though large and small, are, in general, smaller than those for touch and motor conduction, and their function may be unimpaired, if pressure is relieved soon enough.

The properties described above hold only for sudden forceful pressure on a nerve. In the case of gradual or intermittent pressure, the stimulus may not be sufficient at any time to excite the nerve tissue and its presence may remain undetected until paresis develops. This is not infrequently the case in gradually developing gibbus consequent to tuberculosis.

Quite different is the pain produced by stimuli within deep skeletal structures (ligaments, fascia, muscle and periosteum). Here the pain is

vaguely localized, aching in character, radiating over great distances, slow in onset and long in duration. When severe it is often accompanied by sweating, nausea and vomiting. The patterns of radiation are consistent for one point of stimulation, but the extent of radiation depends on the intensity of stimulation. Experimental data have been assimilated to show that the pattern of radiation from one individual to another is fairly consistent. Although these "sclerotomes" appear to have a segmental representation, they do not follow the patterns of corresponding dermatomes The sclerotome and dermatome coincide more nearly in the midportion of the trunk than they do in the extremities.

The pain thresholds of different somatic structures are variable. The periosteum is most sensitive, followed by ligaments and fibrous joint capsules, tendons, fascia and muscle in that order. Ligaments and capsules are particularly sensitive at points close to their osseous attachments Bone for the most part is insensitive, although its endosteal surface apparently contains some pain perceptive nerves



the same region when hypertonic saline is injected into the ... ligament (////). (Redrawn from Kellgren, courtesy of Clin. Science)

... to understand the difference between this "deep" pain ... y demon- ... ueezing a ... ent is not

... ation of pain source Thus stimulation of periosteum ... by ... the ... ince spine is poorly localized and diffuse with radiation to ... from the source.

In this respect another point to be stressed is that a stimulus in any one portion of a segmental sclerotome may give rise to pain which for the subject will not vary though the stimulus be applied at different points Thus Kellgren[14] has shown that pain provoked from the testis, abdominal obliques, and multifidus (1st lumbar segment) was of a similar pattern and character, though usually a little more intense near the site of stimulation Further observations by Kellgren and Lewis[16] have shown that the sclerotomal pain is accompanied by superficial tenderness and muscle rigidity. Clinicians are familiar with these phenomena in the case of visceral disease, but much less so in the case of skeletal lesions

II. HISTORY AND EXAMINATION

Medical History.—A patient who consults the physician because of a sudden twist of the spine with subsequent low back pain does not present a diagnostic problem from the point of view of cause and effect However, in a great many cases, certainly a majority, the pain is spontaneous in onset and a carefully taken history will help greatly to ascertain the cause

First of all a general appraisal of the patient is necessary This will [illegible] ions [illegible] social [illegible] type [illegible] its in [illegible]

[illegible]

ment and previous treatment), the other, with the character (hronologically, one should ascertain when and where the pain began and was its onset associated with injury or illness? Has the pain remained the same, gotten worse or better? Is it intermittent or have there been periods of amelioration or exacerbation? Has there been any change in the site of pain, or has it extended to involve hitherto painless areas? Have there been aggravating incidences since its onset and if so what? Has there been any kind of treatment, self-inflicted or otherwise and with what effect?

[illegible] ous methods [illegible] and should be ap- [illegible] to work, confinement to bed and sleeplessness

2. *Quality.*—This is also variable and depends much on previous experience. Therefore its usefulness is somewhat questionable However, one should differentiate somatic from nerve root pain in so far as possible (See page 1029)

3. *Localization* —This is most important though it is often hard for the patient to situate his pain accurately A point of source will usually be recognized Distal radiation is often present The extent of radiation in many [illegible]

The [illegible]

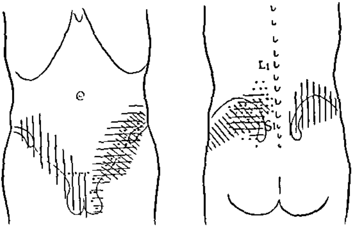
root [illegible]

teristic of irritation of skeletal structures

4. *Duration* —Here is determined whether the pain is steady in duration or intermittent. If it is steady, is it at all times the same or does it have

vaguely localized, aching in character, radiating over great distances, slow in onset and long in duration When
sweating, nausea and vomiting. The
for one point of stimulation, but the
intensity of stimulation. Experimental data have been assimilated to show that the pattern of
consistent. Although t
representation, they do
tomes. The sclerotome and dermatome coincide more nearly in the midportion of the trunk than they do in the extremities.

The pain thresholds of different somatic structures are variable. The periosteum is most sensitive, followed by ligaments and fibrous joint capsules, tendons, fascia and muscle in that order Ligaments and capsules are particularly sensitive at points close to their osseous attachments. Bone for the most part is insensitive, although its endosteal surface apparently contains some pain perceptive nerves.



somatic structures
n the testis (||||),
ness is produced in
umbar interspinous

It is important to understand the difference between this "deep" pain and the "superficial" pain of skin The difference can be readily demon-

the deep pain elicited. From experimental evidence it would appear that the more superficially situated deep structures show fairly accurate localization of pain source. Thus stimulation of periosteum overlying the subcutaneous surface of the tibia can be pointed out relatively accurately by the subject. However, pain from deeply situated periosteum as in the spine is poorly localized and diffuse with radiation to considerable distance from the source.

In this respect another point to be stressed is that a stimulus in any one portion of a segmental sclerotome may give rise to pain which for the subject will not vary though the stimulus be applied at different points Thus Kellgren[14] has shown that pain provoked from the testis, abdominal obliques, and multifidus (1st lumbar segment) was of a similar pattern and character, though usually a little more intense near the site of stimulation Further observations by Kellgren and Lewis[16] have shown that the sclerotomal pain is accompanied by superficial tenderness and muscle rigidity *Clinicians are familiar with these phenomena in the case of visceral disease, but much less so in the case of skeletal lesions*

II. HISTORY AND EXAMINATION

Medical History.—A patient who consults the physician because of a sudden twist of the spine with subsequent low back pain does not present a diagnostic problem from the point of view of cause and effect However, in a great many cases, certainly a majority, the pain is spontaneous in onset and a carefully taken history will help greatly to ascertain the cause

First of all a general appraisal of the patient is necessary This will [illegible] itions [illegible] social [illegible] e type [illegible] ints in [illegible]

[illegible] onset associated with injury or illness? Has the pain remained the same, gotten worse or better? Is it intermittent or have there been periods of amelioration or exacerbation? Has there been any change in the site of pain, or has it extended to involve hitherto painless areas? Have there been aggravating incidences since its onset and if so what? Has there been any kind of treatment, self-inflicted or otherwise and with what effect?

The character of the pain may be discovered by various methods

1. *Severity.*—This is an individ[illegible] factor and should be approached with caution [illegible] inability to work, confinement [illegible]

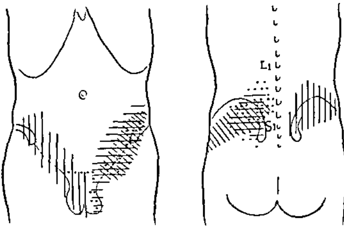
intensity of stimulation. Experimental data have been assimilated to show that the pattern of radiation from one individual to another is fairly consistent. Although these "sclerotomes" appear to have a *segmental* representation, they do not follow the patterns of corresponding dermatomes. The sclerotome and dermatome coincide more nearly in the mid-portion

T

peri

cap

are particularly sensitive at points close to their osseous attachments Bone for the most part is insensitive, although its endosteal surface apparently contains some pain perceptive nerves.



somatic structures
in the testis (||||),
ness is produced in
lumbar interspinous

It is important to understand the difference between this "deep" pain and the "superficial" pain of skin. The difference can be readily demon-

the deep pain elicited. From experimental evidence it would appear that the more superficially situated deep structures show fairly accurate localization of pain source. Thus stimulation of periosteum overlying the subcutaneous surface of the tibia can be pointed out relatively accurately by the subject. However, pain from deeply situated periosteum as in the spine is poorly localized and diffuse with radiation to considerable distance from the source.

If in the history any points of suspicion have come up relative to other systems, these should be investigated before proceeding with the specific examination of the spine.

Stance and Posture.—The spine is first viewed posteriorly. It should be noted whether the iliac crests are level, whether the spine is straight or curved and whether there is a list to one side or the other. In the human being there is a strong reflex to keep the head centered over the feet and the eyes level. Thus, in the normal individual a deviation of the spinal column from the vertical is compensated by an opposite deviation elsewhere whenever possible From the point of view of the spine, full compensation signifies that the first dorsal vertebra is centered over the sacrum,

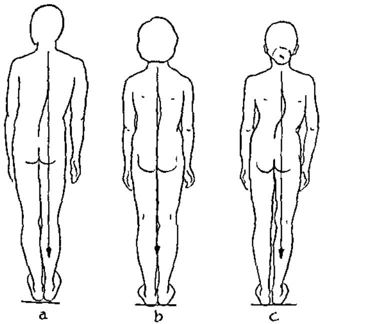


FIG 352—Compensated and uncompensated deviations of posture A plumb line dropped from the 1st dorsal spinal process indicates a right list (*a*), a compensated right dorsolumbar scoliosis (*b*); and an uncompensated right dorsal, left lumbar scoliosis with right list (*c*)

no matter what the spine does in between If the spine is uncompensated, that is, if the first dorsal vertebra is not centered over the sacrum, the patient is spoken of as having a list A convenient measurement of list is that of dropping a perpendicular from the first dorsal spine and measuring how far to right or left it falls of the gluteal cleft. If the iliac crests are level and a lateral curvature of the spine is present, there is a scoliosis the origin of which is in the spine itself. This may be compensated (D1 over sacrum) or uncompensated (list to right or left) A scoliosis is termed right or left depending on the direction of its convexity (right dorsal scoliosis—dorsal curve convex to right) If the pelvis is tilted to one side,

periods of exacerbation? If it is intermittent, at what times do the pains seem to occur? Is it a night or early morning pain suggesting a joint source? Is it a pain associated with work or with being active, suggesting strain, or does it occur only with certain movements?

5. *Reduplication* —Has the patient found, or can he, during the time of the examination, reduplicate his pain or can it be produced by some tecnhic of examination?

6 *Points of Aggravation*.—Is the pain aggravated by coughing or sneezing, by bending or lifting, and if so, is it reproduced in full or just in part?

7. *Points of Alleviation*.—Is the pain relieved by rest, or by any particular types of medication? Aspirin is often dramatic in relieving the pain of an osteoid osteoma. Is it relieved by manipulation or by gentle activity as very frequently is the case in osteoarthritis?

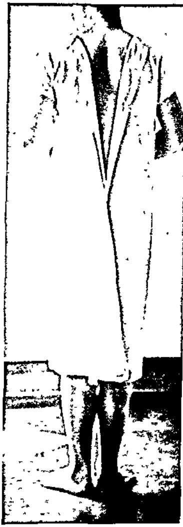


Fig 351 —Examining gown. There is adequate exposure of the back when the edges of the gown are held apart.

Physical Examination.—Once the history has been obtained in full, the physical examination will follow. Needless to say a complete examination is impossible unless the patient is properly prepared. In order to insure the patient's complete cooperation, modesty can be maintained in the case of a man by shorts and in the case of a woman by a short-sleeved gown open and tied at the back There must be enough space and light to allow analysis of stance and gait. A hard straight back chair and a firm examining table are also necessary Examination of the back does not require a great deal of ancillary equipment A tape measure, a reflex hammer, a sharp-pointed pin and sphygmomanometer will usually suffice.

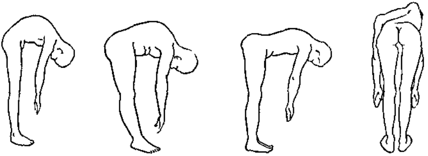
General Appraisal.—The weight and height should be recorded. The patient's general body type is quickly reviewed. In general, it will be sufficient to note whether the individual is stocky and heavy-set with heavy musculature and strong ligaments and tight joints, or whether he is sthenic, tall and lithe, with relatively relaxed joint structure, ligaments and muscles Are the legs well aligned? Beginning with the feet, are these pronated? Are the knees bowed, straight or in some valgus? Are there any abnormal or suggestive skin pigmentations? A cafe-au-lait mark may signify an underlying neurofibromatosis

An analysis of the position in stance is completed by a brief survey from the front Here again list may be noted and the anterior superior iliac spines palpated to check whether the pelvis is level or not. It should be added that slight degrees of curvature of the spine are difficult to detect [illegible]

shoulder blade, or a high shoulder.

Spinal Motions —The spine is now analyzed in movement Again viewing the patient from the back, he is asked to bend forward The lordotic curve of the lumbar spine should first flatten and then reverse slightly as the [illegible]

may suggest some restriction of straight-leg raising on the opposite side



a b c d

Fig 351 —[illegible]
tou [illegible]
ind [illegible]
tha [illegible]
jor [illegible]

If a scoliosis has been detected in the erect position this should again be checked in the flexed position If it is a functional scoliosis, as in the case of compensation for a short leg, it will disappear on bending forward If it is a fixed or structural dorsal scoliosis, it will not disappear In fact, the associated rotation of the vertebrae will produce a prominence of the thorax and overlying scapula on the side of convexity In a lumbar scoliosis the rotation is not so readily evident but can be detected by prominence of the paravertebral muscles on the side of the convexity and a depression of those on the opposite side In cases of very slight degree of scoliosis forward flexion is the best position for detection since the deformity is accentuated When the patient is bent forward it is also wise to view his back from the side. There is usually a smooth curve with its convexity posteriorly extending from the base of the skull to the sacrum If the curve is broken

the normal spine will show a scoliosis with its convexity to the low side. There will be full compensation, if the tilt is not too severe.

The patient is then viewed from the side. The posture is noted. It is normal to stand with a slight degree of lumbar lordosis (anterior curve) and dorsal kyphosis (posterior curve). If these curves are increased or [illegible] muscle spasm in acute low [illegible] lordosis In the sagittal [illegible] very strong tendency to [illegible]tered over the feet. The curves in between will tend to balance one another. Thus, if the dorsal

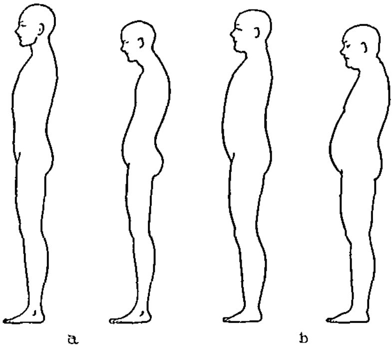


FIG. 353 —Good and bad posture. Posture is best when the vertebral joints are held midway between their extremes of motion It is a position of good balance and requires minimal muscular effort for its maintenance The figures on the left typify good and those on the right bad posture in asthenic (*a*) and sthenic (*b*) body types (Redrawn from Posture and Its Relationship to Orthopedic Disabilities, Posture Committee of the American Academy of Orthopædic Surgeons, Chicago, Ill, 1947)

kyphosis is increased, the lumbar lordosis is usually increased. Sometimes, such a state of affairs does not exist and in order to balance an increased dorsal kyphosis, the patient will stand with a posterior over-carriage, that is, leaning backwards Over-carriage is to the sagittal plane, what list is to the transverse.

The degree of forward inclination of the pelvis should be noted. Is the abdomen pendulous and is the chest well developed or does the patient stand with a very narrow anteroposterior thoracic diameter? There is a tendency for the sthenic body type to have a narrow chest and for the heavy body type to have just the opposite.

are then recorded using the bony landmarks of the anterior superior iliac spine and medial malleolus. In the presence of a fixed deformity of one hip or the other the measurement loses some of its significance, and to be accurate the lower extremities must have equivalent relationships to the pelvis

Hip Motions.—The hip movements are now analyzed. Flexion contracture is noted by maximally flexing the opposite hip to flatten the lumbar lordosis and measuring the degree of the angle between the horizontal surface of the table and the maximally extended extremity The movements of each hip in abduction, adduction, internal and external rotation should be measured. The arcs of rotation are usually most easily obtained rotating the foot and leg inward (external rotation), and outward (internal rotation) with the hips flexed to 90°.

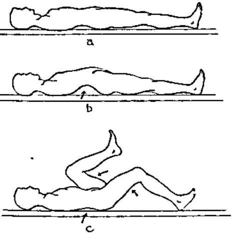


FIG. 356.—[illegible] ...ates the true situation

Passive Spinal Movements.—Movements of the lumbar spine are now performed passively by grasping both legs with the knees bent, and flexing the spine maximally. Keeping the hips flexed to 90° the pelvis is then inclined laterally and then rotated on the spine Note is made if these motions produce pain

Palpation and Tenderness.—The patient is next asked to lie prone with a pillow [illegible] ness of the pillow should be sufficient [illegible] wer spine and structures of the back [illegible] spasm of paravertebral muscles is noted At all times the patient must be fully relaxed Points of tenderness are recorded Just as in examination of the abdomen, the anatomical structures under the examining finger must constantly be kept in mind

and becomes more sharply angular at any point it should be recorded. This is very often the case in structural round back (*i.e.*, "epiphysitis" of the spine).

The degree [illegible]
compamed by
does not incre
upright positio
other. The normal will show an equivalent degree of bending to both sides with a smooth curve starting at the sacrum. Limitation of motion can be detected in one of two ways The maximum degree of motion may not be as much to one side as to the other, or the curve on bending to one side may be broken and begin at a higher level than it does to the other side.

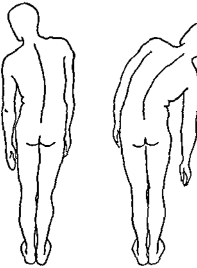


FIG 355 —Lateral bending Motion to the left is restricted in this case It is normal on bending to the right Notice how much higher the curve begins in the left hand figure.

In examination of the lower back, the motions of flexion, extension and lateral bending, are those most important to analyze However, it is [illegible] the patient should be asked to [illegible] This [illegible] cervical lordosis and cervical motions should also be noted The findings are [illegible] normally the lordosis will flatten [illegible] disappear [illegible] d to lie supine on the examining [illegible]

points above and below the patellæ should be made and recorded [illegible] there is atrophy, it may be due to disuse or to neuromuscular disease. The patient is next carefully positioned so that the pelvis is level and the hips are placed in a neutral degree of abduction and adduction. The leg lengths

Rectal Examination—A rectal examination is usually advisable and especially in those cases with coccygeal pain The coccyx may be grasped

region of the sacrosciatic notch can often be reached and bimanual palpation of this region is possible The rectal examination is completed with palpation of the pelvic viscera.

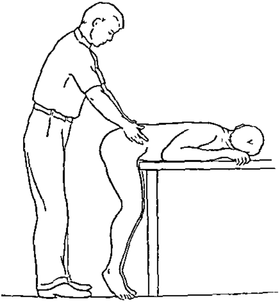


FIG 358—Percussion test With the patient positioned as above the interlaminal spaces are opened as much as possible In the case of irritation of a nerve root, sharp percussion or deep pressure may reproduce the sciatic distribution of pain This is especially true in the presence of disc herniation since the nerve root is forced posteriorly in the spinal canal This position is also a good one for palpation of the back

Neurological Examination.—Neurologically speaking, it is important to analyze the knee and ankle jerks The best position for eliciting the knee jerks is one of sitting with the legs hanging free, that for the ankle jerks is one of kneeling on a chair with
out from the edge of the seat.
by the patient contracting the
locked together. If the reflexes are tested carefully in this fashion small discrepancies will be of much greater significance than if the testing is done casually Other reflexes may be elicited, but are not usually necessary The abdominal and cremasteric reflexes are readily obtained in the supine position, and the sole of the foot may be scratched in order to test for a Babinski response. In the prone position the semi-membranosus tendon jerk may be elicited by a sharp tap of the percussion hammer against the

The spine of each lumbar vertebra is pressed and the interspaces between are palpated. Useful landmarks are the iliac crests; a horizontal from these usually runs across the fourth lumbar process, and the posterior superior iliac spines which lie opposite the second sacral spine. The sacrosciatic notch is palpated where tenderness may indicate irritation of one or more roots of the sciatic nerve. The thighs and calves must also be palpated in any case where there is extension of pain into the legs. The region of the trochanters should also be examined

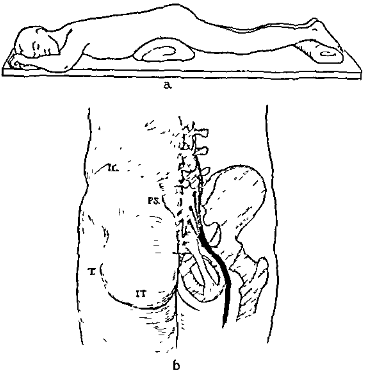


FIG 357.—Palpation of the back. *a* Good positioning is necessary for adequate examination. When the lumbar spine is flat, the bony elements are more easily distinguished from one another The interspinous and interlaminal spaces are opened *b* Useful landmarks include the iliac crests (IC), the posterior superior iliac spine (PS), the greater trochanters (T) and the ischial tuberosities (IT) The position of the sacrosciatic notch and course of the sciatic nerve should always be kept in mind The lumbar roots are shielded from palpation in their extraspinal course.

Percussion Tenderness.—Pressure or percussion over the interlaminal spaces, may, in the cases of disc herniation, produce sharp lancinating pain radiating into the lower extremities Since in the position of extension

lordosis reversed If this cannot be achieved in the prone position with a pillow under the abdomen, the patient may be asked to bend over the examining table. He can usually do this despite the presence of pain, if the knees are flexed and the weight is borne upon the abdomen.

name of one man performed differently not only in different sections of the country but also differently by various clinics in one city. A great many of the tests still have some usage but do not properly belong in routine back examination The following ones described have stood the test of time and should be included in the routine examination of the back

Straight Leg Raising Test.—This is without doubt one of the most useful maneuvers in analysis of low back disorders There is seldom a case of disc herniation that is not accompanied by restricted straight leg raising

The maneuver does two things First, it stretches the roots of the lumbosacral plexus by putting tension on the sciatic nerve, a tension that increases as the leg is raised higher. Secondly, it flattens the lumbar spine by putting tension on the ligaments and extensor muscles of the hip and thigh as they pass from the pelvis to the femur and tibia Thus, a nerve root lesion is not prerequisite for painful or restricted straight leg raising. On the other hand, it is quite true that the restriction of straight leg raising

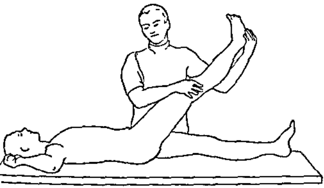


FIG 360 —Straight leg raising The leg being tested should be utterly relaxed and resting in the examiner's hand The knee is fully extended and kept from flexing by gentle pressure against the patella

is usually much more marked in lesions affecting the nerve roots than it is in purely skeletal ones. The angle of straight leg raising, which is limited by tightness of the hamstring muscles is normally less in sthenic than it is in asthenic individuals

The test [illegible]
be ma[illegible]
from t[illegible]
import[illegible] but the patient should also be asked to describe the extent and radiation of the pain. In nerve root lesions, the typical pattern of radiation may be reproduced

This test often bears the name of Lasegue, since it was he who first realized its importance in sciatica Actually his test, as well as Goldthwaite's test are done differently from the method here described

Jugular Compression (Naffziger[19])—This test should be performed wherever there is suspected involvement of the spinal cord or nerve roots It is performed by placing a blood pressure cuff about the neck and inflating

examining thumb which has been placed over its tendon. A similar response may be obtained from the gluteus maximus muscle with the finger placed directly over the center of the muscle

Sensory examination is performed with the point of a pin and light stroke of the finger. Significant zones of sensory disturbance will be found in this manner, and if so the examination should be repeated in more detail, especially in the presence of a clinical picture suggesting nerve root involvement.

An analysis of motor function is done by testing the strength of muscle action in various planes For practical purposes examination may be confined to the foot unless some previous point of history or physical

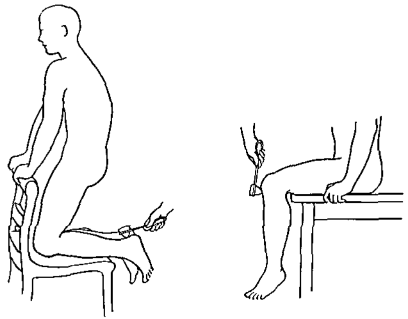


FIG. 359 —Suitable positions in which to elicit the ankle and knee jerks. (Redrawn from Spurling and Thompson, Army Medical Bull)

examination leads one to suspect nerve root pathology The power of dorsiflexion of the great toe (extensor hallucis longus and brevis), of the lateral four toes (extensor digitorum longus), and of the entire foot (tibialis anticus) should be analyzed and compared on the two sides Peroneal function is next tested by the power of eversion of the foot against resistance of the examiner's hand. This again is compared on the two sides Similarly the force of inversion and plantar flexion of the foot and of the toes can be tested.

Special Tests.—*There remain only the various special tests.* Unfortunately, most of these have been described by their originators as diagnostic of one condition or another. That this is not so, is amply attested by the fact that many of them have been described, used and discarded. A complicating factor is that most of these tests have been recognized by the names of their originators. It is not unusual to find a test bearing the

name of one man performed differently not only in different sections of the country but also differently by various clinics in one city. A great many of the tests still have some usage but do not properly belong in routine back examination. The following ones described have stood the test of time and should be included in the routine examination of the back.

Straight Leg Raising Test.—This is without doubt one of the most useful maneuvers in analysis of low back disorders. There is seldom a case of disc herniation that is not accompanied by restricted straight leg raising.

The maneuver does two things. First, it stretches the roots of the lumbosacral plexus by putting tension on the sciatic nerve, a tension that increases as the leg is raised higher. Secondly, it flattens the lumbar spine by putting tension on the ligaments and extensor muscles of the hip and thigh as they pass from the pelvis to the femur and tibia. Thus, a nerve root lesion is not prerequisite for painful or restricted straight leg raising. On the other hand, it is quite true that the restriction of straight leg raising

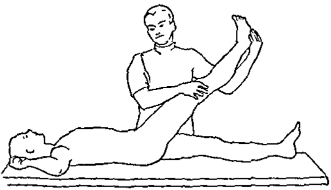


FIG 360.—Straight leg raising. The leg being tested should be utterly relaxed and resting in the examiner's hand. The knee is fully extended and kept from flexing by gentle pressure against the patella.

... asthenic individuals.

The test is performed passively by the examiner and the patient must be made to relax. The knee is held extended and the leg is raised gradually from the table until the point of pain is reached. Not only is this point important to record but the patient should also be asked to describe the extent and radiation of the pain. In nerve root lesions, the typical pattern of radiation may be reproduced.

This test often bears the name of Lasegue, since it was he who first realized its importance in sciatica. Actually his test, as well as Goldthwaite's test are done differently from the method here described.

Jugular Compr...

wherever

It is perf...

examining thumb which has been placed over its tendon. A similar response may be obtained from the gluteus maximus muscle with the finger placed directly over the center of the muscle.

Sensory examination is performed with the point of a pin and light stroke of the finger. Significant zones of sensory disturbance will be found in this manner, and if so the examination should be repeated in more detail, especially in the presence of a clinical picture suggesting nerve root involvement

An analysis of motor function is done by testing the strength of muscle action in various planes. For practical purposes examination may be confined to the foot unless some previous point of history or physical

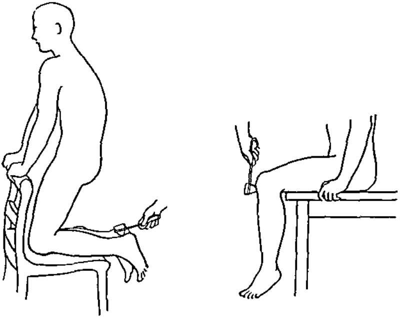


FIG 359 —Suitable positions in which to elicit the ankle and knee jerks (Redrawn from Spurling and Thompson, Army Medical Bull)

examination leads one to suspect nerve root pathology. The power of dorsiflexion of the great toe (extensor hallucis longus and brevis), of the lateral four toes (extensor digitorum longus), and of the entire foot (tibialis anticus) should be analyzed and compared on the two sides. Peroneal function is next tested by the power of eversion of the foot against resistance of the examiner's hand This again is compared on the two sides. Similarly the force of inversion and plantar flexion of the foot and of the toes can be tested.

Special Tests.—There remain only the various special tests. Unfortunately, most of these have been [illegible] diagnostic of one condition or another. T
fact that many of them have
complicating factor is that most of these tests have been recognized by the names of their originators. It is not unusual to find a test bearing the

name of one man performed differently not only in different sections of the country but also differently by various clinics in one city A great many of the tests still have some usage but do not properly belong in routine back examination. The following ones described have stood the test of time and should be included in the routine examination of the back

Straight Leg Raising Test.—This is without doubt one of the most useful maneuvers in analysis of low back disorders. There is seldom a case of disc herniation that is not accompanied by restricted straight leg raising

The maneuver does two things. First, it stretches the roots of the

root lesion is not prerequisite for painful or restricted straight leg raising. On the other hand, it is quite true that the restriction of straight leg raising



FIG 360.—Straight leg raising The leg being tested should be utterly relaxed and resting in the examiner's hand The knee is fully extended and kept from flexing by gentle pressure against the patella

in asthenic individuals

The test is performed passively by the examiner and the patient must be made to relax The knee is held extended and the leg is raised gradually from the table until the point of pain is reached Not only is this point important to record but the patient should also be asked to describe the extent and radiation of the pain. In nerve root lesions, the typical pattern of radiation may be reproduced.

This test often bears the name of Lasegue, since it was he who first realized its importance in sciatica Actually his test, as well as Goldthwaite's test are done differently from the method here described

Jugular Compression (*Naffziger*[19]).—This test should be performed wherever there is suspected involvement of the spinal cord or nerve roots It is performed by placing a blood pressure cuff about the neck and inflating

examining thumb which has been placed over its tendon A similar response may be obtained from the gluteus maximus muscle with the finger placed directly over the center of the muscle.

Sensory examination is performed with the point of a pin and light stroke of the finger. Significant zones of sensory disturbance will be found in this manner, and if so the examination should be repeated in more detail, especially in the presence of a clinical picture suggesting nerve root involvement.

An analysis of motor function is done by testing the strength of muscle action in various planes. For practical purposes examination may be confined to the foot unless some previous point of history or physical

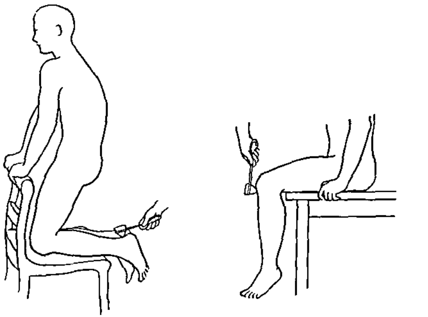


FIG 359 —Suitable positions in which to elicit the ankle and knee jerks. (Redrawn from Spurling and Thompson, Army Medical Bull)

examination leads one to suspect nerve root pathology. The power of dorsiflexion of the great toe (extensor hallucis longus and brevis), of the lateral four toes (extensor digitorum longus), and of the entire foot (tibialis anticus) should be analyzed and compared on the two sides Peroneal function is next tested by the power of eversion of the foot against resistance of the examiner's hand. This again is compared on the two sides Similarly the force of inversion and plantar flexion of the foot and of the toes can be tested.

Special Tests —There remain only the various special tests Unfortunately, most of these have been described by their originators as diagnostic of one condition or another. That this is not so, is amply attested by the fact that many of them have been described, used and discarded. A complicating factor is that most of these tests have been recognized by the names of their originators. It is not unusual to find a test bearing the

name of one man performed differently not only in different sections of the country but also differently by various clinics in one city. A great many of the tests still have some usage but do not properly belong in routine back examination. The following ones described have stood the [illegible]

disc herniation that is not accompanied by restricted straight leg raising. The maneuver does two things. First, it stretches the roots of the lumbosacral plexus by putting tension on the sciatic nerve, a tension that increases as the leg is raised higher. Secondly, it flattens the lumbar spine by putting tension on the ligaments and extensor muscles of the hip and thigh as they pass from the pelvis to the femur and tibia Thus, a nerve root lesion is not prerequisite for painful or restricted straight leg raising. On the other hand, it is quite true that the restriction of straight leg raising

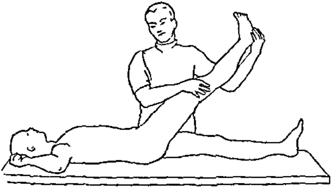


FIG 300 —Straight leg raising The leg being tested should be utterly relaxed and resting in the examiner's hand The knee is fully extended and kept from flexing by gentle pressure against the patella

is usually much more [illegible]
in purely ske[illegible]
by tightness [illegible]
in asthenic individuals

The test is performed passively by the examiner and the patient must be made to relax. The knee is held extended and the leg is raised gradually from the table until the point of pain is reached Not only is this point important to record but the patient should also be asked to describe the extent and radiation of the pain In nerve root lesions, the typical pattern of radiation may be reproduced

This test often bears the name of Lasegue, since it was he who first realized its importance in sciatica Actually his test, as well as Goldthwaite's test are done differently from the method here described

Jugular Compression (*Naffziger*[19]) —This test should be performed wherever there is suspected involvement of the spinal cord or nerve roots It is performed by placing a blood pressure cuff about the neck and inflating

it to 40 mm of mercury and holding it there for the *length of one minute* By this method cerebrospinal fluid pressure is increased The patient is asked to indicate when and if his pain is reproduced and if it is reproduced how accurately so When the exact pattern of the patient's leg pain is

A negative

. described as pathognomonic of sacroiliac disease, we believe that the test of iliac compression is the most sensitive. It is performed most easily with the patient *lying on his side* The examiner applies firm pressure against the uppermost *iliac crest* When positive, pain will be produced in the region of the involved sacroiliac joint.

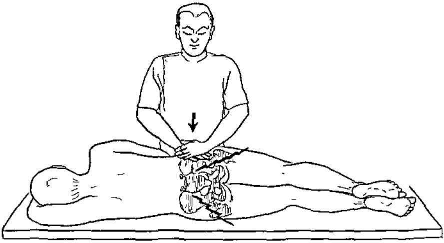


FIG 361 —The iliac compression test. The position of lateral recumbency is the most satisfactory in which to elicit the pain of sacroiliac disease. Compression of the joint is produced by downward pressure of the examiner's hands

Passive Extension Test (Duncan-Hoen[7]).—This test is performed with

vel

the

spi

patient experiences spontaneously it is highly suggestive of a herniated disc with nerve root impingement A great many lesions of the back are associated with pain on hyperextension

finished As can be seen, it requires con-

In acute cases it will not be possible to

However, it will often be found that after a day or two of rest and sedation, a more satisfactory analysis can be carried out.

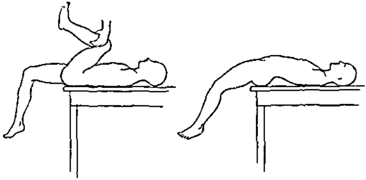


Fig 362 —The passive extension test Pain is produced by exaggeration of the lumbar lordosis as the legs are allowed to hang over the end of the table. It is relieved by flexion of one or both hips



Fig 363 —Xrays of normal spine (see Fig 365 for legend) AP and Lateral x-rays of lumbar spine—see p 1044 of text for technique Note good detail of most of lumbar spine but obscurity of lumbosacral region.

Legend

B—body
D—disc space
I—isthmus or pars interarticularis
IF—inferior facet
IVF—*intervertebral foramen*
L—lamina
P—pedicle
PM—psoas margin
S—spinous process
SF—superior facet
SI—sacroiliac joint
T—transverse process
IPD—interpedicular diameter

X-ray Examination.—A routine examination of the back is not complete unless x-rays have been made. To the trained observer, an A.P. and lateral view of the entire lumbar spine and sacrum is all that is usually needed. First A.P. and lateral views are made of the lumbar spine with the x-ray tube centered at the upper lumbar region and inclined slightly distally (10°). A true or 45° A.P. of the lumbosacral region is then obtained. For this view the direction of the tube is adjusted to the inclination of the sacrum. The actual angle of the tube is usually nearer 30° than 45°. The tube itself is centered opposite the third or fourth lumbar vertebra. If the technique is good, detail of the vertebral bodies, sacrum, sacroiliac and intervertebral joints should be good enough for the detection of significant abnormalities including defects of the neural arch and facets.



FIG. 364.—"45° AP" and Lateral x-rays of lumbosacral level and sacrum. Note good detail of 5th lumbar vertebra, sacrum, lumbosacral disc space and sacroiliac joints.

Special roentgenographic views are used only to obtain better detail of spinal parts or to detect the presence of specific pathologic processes. [illegible] lesions of the neural [illegible] is difficult, how-

[illegible]

the right and then [illegible]

[illegible]

the lumbosacral spine in full flexion a[illegible] en[illegible]
disease when abnormal motion is det[illegible]



FIG 365 —Oblique x-ray of lumbar spine Note good detail of facets and pars interarticularis (See Fig 363 for legend).

X-ray [illegible]
unless *x*-
view of
First A P. and lateral views are made of the lumbar spine with the *x*-ray tube centered at the upper lumbar region and inclined slightly distally (10°) A true or 45° A.P. of [illegible] For this view the direction of th [illegible] of the sacrum The actual angle of [illegible] The tube itself is centered opposite the third or fourth lumbar vertebra. If the technique is good, detail of the vertebral bodies, sacrum, sacroiliac and intervertebral joints should be good enough for the detection of significant abnormalities including defects of the neural arch and facets.



FIG 364 —"45° AP" and Lateral *x*-rays of lumbosacral level and sacrum Note good detail of 5th lumbar vertebra, sacrum, lumbosacral disc space and sacroiliac joints

Special roentgenographic views are used only to obtain better detail of spinal parts or to detect the presence of specific pathologic processes Oblique views show the intervertebral foramina and lesions of the neural arch and facets exceptionally well The *x*-ray technique is difficult, however. A P A. film made with the patient standing and bending first to the right and then to the left is often helpful in diagnosing the level of a disc lesion If the restriction of lateral movement is diffuse throughout the lumbar spine, the *x*-rays are of no help but if the restriction of lateral bending seems to be confined to one disc space and to one side only, it is indicative of pathology at this level (Duncan-Hoen[7]). Lateral films of the lumbosacral spine in full flexion and extension indicate the level of disease when abnormal motion is detected.



FIG 365.—Oblique x-ray of lumbar spine Note good detail of facets and pars interarticularis (See Fig 363 for legend).



FIG 366.—Lateral bending films PA x-rays of lumbar spine in left (a) and right (b) bending In a the entire spine bends to the left and all the interspaces are wider on the right than on the left In b the 4th lumbar space (arrow) does not close on the right indicating disc protrusion The fifth lumbar disc space is not well visualized, but there is usually little lateral motion at this level

Pantopaque Myelogram.—This procedure should be reserved for obscure cases where an intraspinal lesion is considered a possibility. The technique is arduous for the patient and occasionally it is difficult to remove all the oil. There are reports of arachnoiditis in the presence of retained pantopaque. At the time of pantopaque examination, if done, the manometrics of the spinal fluid should be measured to detect the presence of a block. Spinal fluid should also be withdrawn for laboratory analysis. An erythrocyte and leukocyte count with differential, a determination of the protein level and Wassermann should be done.



FIG. 367.—Pantopaque myelogram *a* There is normal filling of the lower end of the dural sac. The axillary pouches (nerve root sheaths) are well visualized at the lumbosacral level (arrow) In order to complete the examination each intervertebral [illegible]

Laboratory Examination.—As a routine, it has been our practice to obtain a urinalysis, complete blood count and erythrocyte sedimentation rate on all patients. When elevated, we have used the sedimentation rate as an indication for further study of the patient. It is usually not elevated in the presence of a purely mechanical lesion or a disc herniation. Other laboratory tests are only indicated when specific disease processes are suspected

III. LOW BACK OR LUMBOSACRAL STRAIN

Physicodynamics.—In order to understand the principles of strain in relation to causation of low back pain, one must reconsider for a moment the physiology of the spine in relation to stance, movement and work.

It has already been pointed out that under normal conditions the spine is well balanced in the erect position. The head is centered over the feet and the successive spinal curves neutralize one another. The ligaments, assisted by the superincumbent weight of the body, exert a contractile force which is counterbalanced by the inherent expansile turgor of the discs. In addition the intervertebral joints are held midway between their extremes of motion. A cushioning zone is thus provided in which the muscles may first provide a buffering action and the ligaments finally a checking action. The normal spine, therefore, is a resilient structure, prepared amazingly well for the shocks and excessive pressures to which it is continually subject.

The normal physiological state can be upset, however, by faulty posture or disease. If posture is poor, the intervertebral joints lie near one extreme of motion. The muscles and ligaments are shortened on one side and lengthened on the other. The *buffering mechanism is markedly impaired.* If by specific disease processes, or by the degenerative effects of wear and tear, the resiliency of the discs is lost, the spine can no longer respond efficiently to sudden disturbances of its equilibrium. Thus any and every pathological change that may occur will leave its mark upon the spine and it becomes more prone to injury.

Very few people are aware of the tremendous mechanical forces that are at play in the spine during normal daily life. Any movement is associated with changes of pressure that defy calculation, but these are as nothing when compared to the leverage forces produced by such acts as sneezing, *bending or lifting. It requires* very little knowledge of physical laws to understand that these pressures are greatest in the lower lumbar region and it is here that one finds the most marked signs of wear and tear.

Putting all these facts together, one can understand how overstrain of the back occurs so easily, and in the presence of such trivial initiating

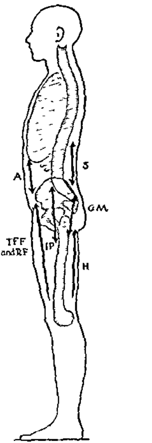


FIG 368 —The important muscles in relation to posture of the lumbar spine Contraction of the abdominals (A) and gluteus maximus (GM) reduces the lumbar lordosis by flexion of the lumbar spine and by reducing the forward tilt of the pelvis Contraction of the other muscles increases the lordosis The rectus femoris (RF), the tensor fascia femoris (TFF), and the iliopsoas (IP) flex the hip and increase the forward tilt of the pelvis. The sacrospinalis (S) extends the spine and thereby increases the lumbar lordosis. The hamstrings (H) are also illustrated since they frequently become shortened in cases of low back pain and sciatica

injuries. In many instances the pain of overstrain may seemingly appear spontaneous, but it is the summation of inherent degenerative processes with repeated subclinical traumata that brings the pathological lesion to clinical significance

Pathogenesis.—What is the pathology of strain? Unfortunately this is a hard question to answer since it is seldom that fresh material is available for examination. Much of it occurs in the disc and will be subsequently described (p 1060). There are acute tears of ligaments, muscles and fascia Occasionally there are avulsion fractures as of the transverse or spinous processes However, of greater significance, are the less violent reactions, such as the wearing of cartilaginous joint surfaces, the minor microscopic tears of ligaments and muscles; and the gradual fibrosis of the various specialized connective tissues. To support these contentions there are the studies of the aging process in somatic tissues,[6] the occasional acute

and progress to advanced stages, with little or no clinical evidence. Furthermore, x-rays are often entirely negative, or the changes seen of equivocal significance.

FACTORS PREDISPOSING TO INSTABILITY OR STRAIN

Faulty Posture.—As is widely known, bad posture is relatively prevalent in the human race [15,24] It can be classified into two groups. The first of these is a *functional* one in which the position of faulty posture is correctible. It is extremely common in children and progressively less so in older age groups. The second is a *structural* group in which the deformity is fixed. The cervical lordosis is increased. The dorsal spine is rounded and its motion limited. The exaggerated lordotic lumbar curve is fixed due to shortening of the sacrospinalis muscles The interspinous ligaments are contracted and the tips of the spinous processes may actually be in contact

generative changes by x-ray

In a purely functional state, faulty posture is rarely painful It may cause some fatigue and backache, but it is only when the position is fixed that the symptoms are severe.

Spondylolisthesis.—Generically this term means a slipped vertebra Clinically, it is associated only with forward or anterior displacement of a vertebra upon the one below. (See Fig 370).

Most commonly a defect of ossification is present in the isthmic portion of the vertebral arch, that is in the part lying between the superior and inferior articular processes The defect when present bilaterally results in disconnection of the anterior and posterior halves of the vertebra. The two halves are held together only by fibrous tissue instead of bone. The an-

terior half may slip forward with the superincumbent spine while the posterior half remains behind, and a true spondylolisthesis results. If the defect is present only unilaterally, or is present bilaterally but without forward slip, the condition is termed a **spondylolysis.**[15]

occurring in the act of birth with subsequent fibrous union. However, the

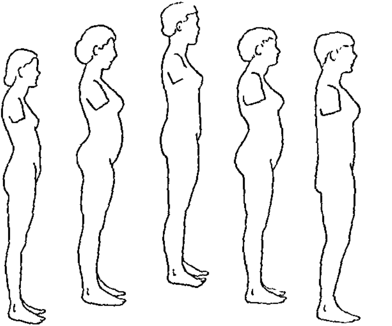


FIG 369.—Various common postural attitudes. From left to right they are round, hollow, good, round hollow and flat. (Redrawn from Posture and Its Relationship to Orthopædic Disabilities. Posture Committee of American Academy of Orthopædic Surgery. Chicago, Ill., 1947.)

condition has been seen in identical twins. Willis[23] found the bilateral defect present in 4 per cent of several hundred skeletal specimens. The lesion is most common in the 5th lumbar vertebra though it can occur at any level of the spine. (See Fig. 371).

There is great variability in the degree of slipping from one case to the next. Sometimes the condition spontaneously becomes symptomatic in childhood, sometimes later in adult life and sometimes never. Very often the symptoms

In such cases the symptoms come on gradually and there is no reason to fear sudden paraplegia under normal conditions.

Diagnosis rests on roentgenographic demonstration of the defects in the neural arch and forward slipping of the vertebra. The treatment of choice in the presence of symptoms is operative fusion, but sometimes conservative therapy is advisable.

[illegible]

...itions are recognized by x-ray, especially in oblique views.

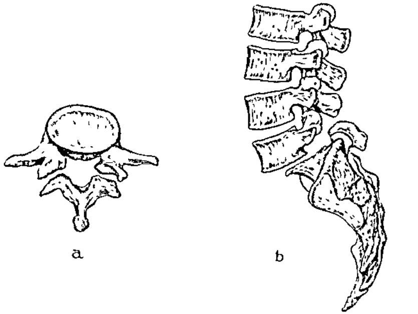


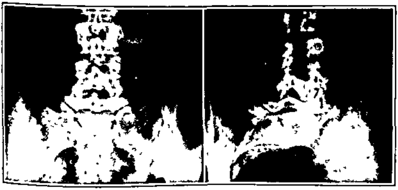
Fig. 370.—Spondylolisthesis. *a.* Vertebra showing failure of ossification of the interarticular portions of the neural arch. Without forward slip of the involved vertebra, this is called spondylolysis. (Willis, courtesy of Jour. Bone & Joint Surg.) *b.* The deformity (spondylolisthesis) which may occur as the result of the above. The defect is in the arch of L5. The posterior portion of the arch including the inferior facets remains with the sacrum while the rest of the 5th lumbar vertebra slips forward, carrying with it, the entire spine. All degrees of slipping are seen.

Trauma.—Each spinal injury that occurs tends to weaken the spine and predispose to further injury, regardless of type and extent of damage. This is especially the case where protection of the injured part is not adequate. Ligaments and muscles heal only by scar formation and the annulus fibrosus is almost powerless to heal tears of its substance. These circumstances, unfortunately, are beyond the control of the physician, but the patient should be warned to protect his back following injury in order to provide adequate time for fibrous repair.

Disc Degeneration.—The pathologic aspects of this condition are discussed later (p. 1060). The intervertebral joint loses stability in the presence of dessication of the disc as the result of loss of turgor in the nucleus, even



Fig 371.—Lateral x-ray showing a Grade I spondylolisthesis The defect in the neural arch is also seen The deformity may occur as the result of abnormalities of the facets or disc degeneration, though the slipping is seldom of this degree



the spine

The instability
For a time the
e instability, but
they are rendered less resistant to overstrain and liable to injury Actual posterior shift (reverse spondylolisthesis) of the upper vertebra on the lower may occur and often the facet joints are telescoped as the ligaments and supporting structures give way The end stage is usually one of marked narrowing of the disc space.

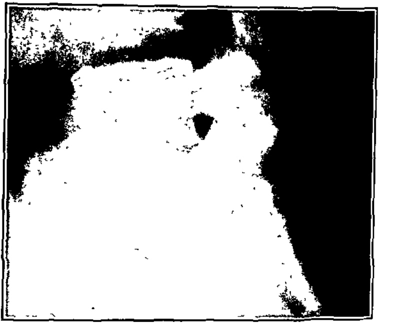


FIG 373 —Retrodisplacement Lateral x-ray of a case with slight narrowing of the 5th lumbar disc and retrodisplacement of the 5th lumbar vertebra on the sacrum This condition is often called reverse spondylolisthesis It is often associated with pain and is seen frequently without narrowing of the disc space It is a strong indication for spine fusion

Stages in the Phylogenetic Shortening of the Spine Which may Predispose to Strain.—The structure of the lowest lumbar vertebra and the first sacral segment is quite variable. Either may take on some or all of the characters of the other. When this happens the vertebræ are spoken of as transitional.

Willis[23] found that the fifth lumbar vertebra was partially sacralized in 4 7 per cent and completely sacralized in 1 5 per cent of nearly 1500 skeletons In the same study he found that the first segment of the sacrum was partially lumbarized in 1.2 per cent and completely lumbarized in 4 1 per cent of the spines.[23] Certainly complete fusion or sacralization of the fifth ... not predispose to strain It is also hard to see how
so.
e, the situation
arge transverse

processes articulating with the sacrum, sometimes on one side only, and at others on both sides It is also in these cases that the facets are often assymetrical, that is, in different planes on the two sides. These changes interfere with normal motion and therefore predispose to strain They are not incompatible, on the other hand, with normal existence, and should never be considered cause for treatment in the absence of symptoms

A word is also necessary about spina bifida occulta This condition is [illegible]

the spinous processes are present as loose ossicles They are embedded in fibrous tissue and it is very unlikely that they produce symptoms in any way.

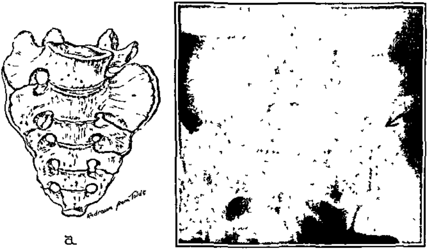


FIG 374 —Articulating transverse process *a* An incompletely sacralized fifth lumbar vertebra is shown with an articulating transverse process on the right The intervertebral disc is usually congenitally narrow in these cases *b* An articulating transverse process on the left side of the 5th lumbar vertebra The articulation is usually a fibrous one. This congenital anomaly is not uncommon and is not necessarily associated with pain.

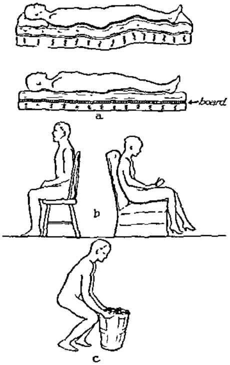
Clinical Picture of Low Back or Lumbosacral Strain.—Low back strain usually occurs in an acute or subacute (chronic) form The acute syndrome is one of sudden onset and usually the consequence of a definite injury, no matter how trivial. The severity of the clinical picture is frequently out of proportion to the nature of the injury The subacute or chronic form, on the other hand, is usually of relatively long standing. There is very often no history of trauma whatsoever. Occasionally acute strain will pass into a subacute stage. Likewise a low grade chronic strain not infrequently becomes exacerbated into a more acute form. This process may recur again and again unless treated properly

Acute Low Back Strain.—The patient is in severe pain The pain is usually diffuse over the lower back and associated with paravertebral muscle spasm. The patient adopts a protective attitude of [illegible]

flexion and perhaps some list. Any motion is painful and associated with aggravation of the spasm.

Complete examination is out of the question and hardly necessary to make the diagnosis The muscles of the back should be examined to see where there is spasm. The point of maximum tenderness should be sought. Testing of the knee and ankle reflexes will serve to rule out major nerve root compression. An x-ray should be obtained at the earliest opportunity to rule out fracture.

The patient should be put to bed, preferably a bed with a hair mattress reinforced by an underlying board (Fig. 375). If such a board is not available, shifting the mattress to the floor will often substitute. Sedation is



given as required *Heat in the form of hot moist packs* should be applied to the back, since it is effective in relieving the muscle spasm. Light massage is often helpful in this respect, but deep massage should be avoided Sometimes novocaine infiltration of the tight muscles is helpful. As the pain subsides more complete examination should be carried out in order to discern any predisposing factor that may be present. As soon as the straight leg raising is free through an arc of 45° or more, the patient may be allowed up. His back must be protected until his symptoms have

subsided completely. If the strain has been severe, a brace or canvas [illegible]

Many acute attacks are not as severe as the one painted above In these cases ambulatory treatment may be sufficient. Adhesive strapping, novocaine injection and physiotherapy including heat and massage are all useful measures of treatment Again the patient should be watched for developing contractures. These should be corrected with exercises whenever present.

Subacute or Chronic Low Back Strain.—This syndrome which is probably the commonest of low back conditions is extremely variable Symptoms can be nothing but a very mild low backache aggravated by bending and

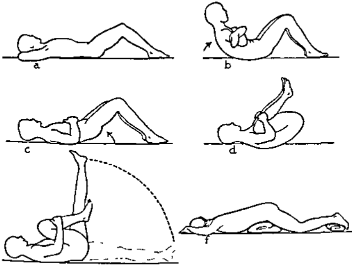


FIG 376—Postural exercises Carefully supervised exercises will do more for the patient with a weak back than almost any other conservative measure They should be graduated carefully and the routine varied as indicated by the patient's response The exercises are exagge[illegible]
is described with each [illegible]
hold chest up and [illegible]

lifting and improved by rest. On the other hand, the pain may be severe in which case the patient is most uncomfortable. Rarely is it severe for more than short periods at a time subsiding in between to a nagging diffuse low backache with radiation into one or both legs It is almost always aggravated by activity and improves with rest.

The physical signs are also quite variable. In the mild cases there may be no limitation of motion, but usually the extremes of motion are painful. There is almost always tenderness at some point over the lower spine. As the severity of the syndrome increases one notes additional signs such as

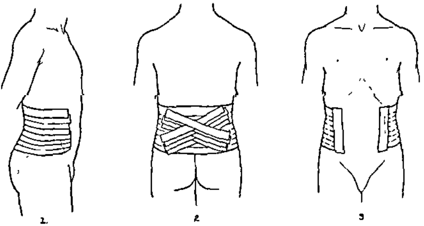


Fig 377 —Strapping of the low back. 1. Overlapping horizontal adhesive strips 2 Diagonal layer of overlapping strips 3 Final layer of horizontal strips with anchoring anterior vertical strips



[illegible] exaggerated Note
r superior iliac spines
s molded well under
is not allowed to sit

partial restriction of straight leg raising, loss of motion, abnormal postural attitudes and muscle spasm particularly in response to movement

X-rays are made The erythrocyte sedimentation test is obtained Pre-
conditions (*see*

which case the
In less severe
indicated The
patient must be instructed in proper postural principles The proper sleeping and sitting positions are shown. He is taught to avoid bending

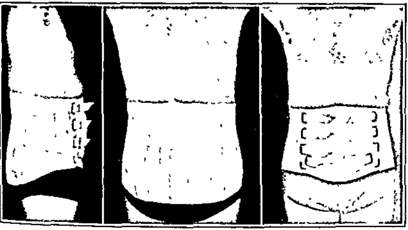


Fig 379 —Lumbosacral belt. Steel stays are incorporated into the back of the support It must extend from the iliac crests to just over the costal margins

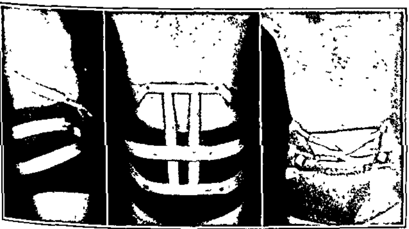


Fig 380 —Goldthwaite brace. A light support that incorporates the principles of good posture The lowest strap should come just over the trochanters and under the anterior superior iliac spines (marked). The vertical steels in back are bent to a position of slight lordosis.

and lifting or methods of doing these acts so as to minimize the pressure on his back. He is instructed in postural exercises, which have as their principle the reduction of lumbar lordosis, the strengthening of the abdominal muscles and the glutei maximii in order to correct the forward tilt of the pelvis, and the stretching of any contracted muscles that are present. Stretching must be done cautiously at first. The groups usually requiring stretching are the sacrospinalis and the hamstrings. Sometimes, a brace

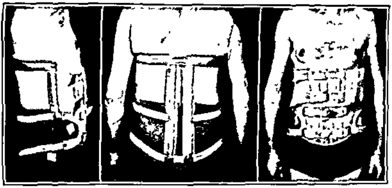


FIG 381.—Butterfly brace. A strong support to provide protection for the painful low back. The loop on each side grasps the anterior superior iliac spines (marked) Flexion of the hip is slightly hindered



FIG 382.—Spine fusion. Spinal fusion is sometimes necessary to relieve low back pain. The 4th and 5th lumbar vertebræ have been united to the sacrum by bone grafts The Wilson metallic plate serves as fixation to prevent motion while the bone is healing Once the fusion is consolidated the plate and bolts may remain without causing harm.

will be necessary at first. In this case, a Goldthwaite or postural brace is indicated. This brace is designed to give adequate support but to flatten the lumbar lordosis. It is a constant reminder to the patient to stand with good posture. In other words the patient must not only follow his postural exercises but he must *live* with good posture If he does not keep up, his

to grow together Techniques for lumbo-sacral fusion are numerous, but unfortunately there are still all too many failures. Since the operation requires a prolonged convalescence a failure is a serious disaster, and for this reason spinal fusion is not entertained lightly. The senior author[26] has devised a technique using a metal plate It has been in use at our clinic for ten years and has provided the best results so far, but still failures occur All in all the decision for spinal fusion will have to be made by the patient and surgeon together. When the patient understands the importance of this step he may sometimes prefer to live with his pain than to go through with surgery.

IV. DEGENERATIVE DISC DISEASE

Herniation of the intervertebral disc as a cause of sciatica did not re-

the terms sciatica and herniated
nomous. Actually there are many other etiologies for sciatica and they must not be forgotten Traumatic, neoplastic, infect

cau

wit

or supporting ligaments by disease may cause pressure on nerve roots as a secondary phenomenon. As already mentioned, referred pain from pathological states in deeply situated skeletal tissues may be confused with the sciatica that results from nerve root irritation

Physiology.—The anatomy of the intervertebral disc has been briefly described, together with the relationship of nerve roots in the lumbar region (pp 1024, 1026–1027) One recalls the concentric lamellæ of the annulus fibrosus which with the cartilage plates enclose the semifluid nucleus pulposus Since liquid substances are relatively incompressible, the nucleus of itself provides little if any cushioning of vertical forces What compressibility there is in the discs must come from elasticity of the annulus as pressure is distributed to all its parts by distortion of the nucleus That the latter exerts a continual expansive force is shown by bulging of the nucleus when a fresh specimen of disc is cut and by the elongation of the spine when its ligaments are cut in such a way as to preserve the integrity of the disc. This pressure is probably exerted by an osmotic force, since there is evidence that the spine elongates slightly when in the horizontal position where interstitial hydrostatic pressure is at a minimum and shortens in the upright The nucleus also shows a

much greater affinity for water than the surrounding annulus in post-mortem specimens.

During motion of the spine the nucleus moves slightly forward on extension and backward on flexion It moves slightly to the opposite side on lateral inclination It also changes shape slightly as the margins of the disc are alternately compressed and elongated during motion.

Pathogenesis.—As one can well imagine, the disc must have to be [illegible]
it
life.
annulus, gradual loss of cartilage cells and increase in fibrosis, gradual loss of the differentiation between nucleus and annulus and dessication and cavitation of the nucleus itself. The disc is only vascularized significantly in the early years of life and repair especially of central lesions is poor at best.

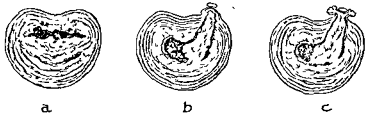


FIG 383 —Types of disc degeneration *a* Degeneration of the disc There is fissuring of the annulus, and the nucleus is loosened and dessicated There is no herniation This is the preliminary phase of degenerative disc disease *b* There is degeneration [illegible]
subsequent *extrusion*

Though the cartilaginous plates are more resistant to inanition, it is not infrequent for them to develop poorly during growth with breaks in their continuity resulting in herniations of the nuclear material into the substance of the vertebral bodies (Schmorl's nodules). These changes are frequently associated with a so-called "epiphysitis" of the spine (*see* page [illegible] also be destroyed by
bone of the vertebra

Thus the intervertebral joint shows the reactions to wear and tear at least as much as if not more than peripheral joints and the term of Degenerative Disc Disease has the same connotation for the spine as De-
[illegible] he spine.
[illegible] ater me-
chanical load as in the lower lumbar spine They are generally progressive with advancing age.

As these degenerative changes take place the normal spinal mechanics are rendered inefficient. This is obvious in the presence of narrowing of

the disc space, but in general thinning of the disc only comes late in the process Before thinning can take place turgor of the nucleus must be lost, the annulus must degenerate, disc material must protrude or be absorbed, and the small posterior articulations must telescope or the interspinous ligaments and spinal muscles stretch or tear. It is in this phase that the back is so vulnerable to ligamentous and muscular strains, and the back (*i e.*, the spinal joints) is said to be **unstable.**



Fig 384 —Phantom nucleus This phenomenon is seen occasionally and is said to be due to a negative pressure produced in the nucleus by sudden distraction of the vertebral bodies However, it may be a sign of fatty degeneration In this case there is some narrowing of the disc as well

Are these degenerative changes in the disc painful by themselves? Probably not until they reach the periphery, since most frequently they represent a response to gradual wear. However, single injuries may produce similar changes or greatly accelerate the process when superimposed on degeneration, and in these cases the changes may be associated directly with pain.

Again, returning to anatomical considerations, one recalls that the spinal nerve roots are intimately related to the discs especially as they pass

ietion of the intraforaminal portion of the nerve root. This is not always the case certainly, since posterocentral protrusions occur as well, but there

is no doubt that the latter are much less common Anterior and lateral herniations are infrequent because of the greater strength of the anterior longitudinal ligaments, and also because the nucleus lies slightly posterior to the central point of the disc. As the disc softens, however, bony osteophytes form anteriorly and laterally reflecting bulge of the disc in these directions.

Herniations may occur in one of a number of ways. There may be a herniation of a small or large part of the disc with complete rupture of the annulus or there may be protrusion without complete rupture of the overlying annular fibers In the former case the disc material is found loose in the spinal canal; in the latter it escapes only after incising the annulus In each instance the nerve root is found stretched over the bulging or herniated material and must be carefully retracted to remove the lesion Very often further degenerated materials will be found within the intervertebral disc space, either loosely attached or lying free If the lesion has occurred recently the nerve root is easily separated from the disc.

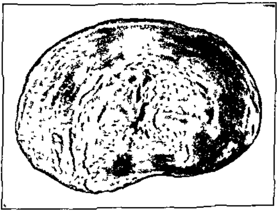


FIG 385 —Disc herniation Transverse section of a disc with central degeneration and with rupture through the posterolateral fibers of the annulus Extrusion has occurred

[illegible]

addition there may [illegible]
nerve root needs to move freely in its canal.

In some complete extrusions one will be surprised at times to find very [illegible] in the canal though the rent in the annulus is clearly [illegible]y from the site of hernia- [illegible] be absor[illegible]

always examine the disc [illegible] material.

There are a good many cases in which the pathological findings are not nearly so distinct In some no evidence of protrusion or extrusion is present. A great many of these represent earlier stages of degeneration in which the disc is softened, but the annulus is not weakened enough to

discs for these cases He
tresses of activity, only to
withdraw on assuming the horizontal position and also during anesthesia. There is little definite pathological ground to confirm this hypothesis, however.

Neuropathological Concepts.—A word is necessary about the neurological aspects of herniation If the herniation is well laterally it will involve the nerve root in the foramen and the disc and nerve root are of the same segment (L5–S1 disc and L5 root) One nerve root is involved and since there is considerable overlap of sensory and motor innervation in the lower extremities there will usually be little or no clinically detectible loss of either of these types of innervation. The most frequent finding is reflex loss or weakness

Very frequently the disc herniation is nearer the midline, but still definitely lateral to it and in these cases the nerve root of the segment below is involved (*i e*, L5–S1 disc and S1 nerve root) Here again one nerve root only is involved and the neurological findings are confined essentially to reflex loss. Rarely the disc herniation is in the midline where the spinal canal is large and the roots of the cauda equina are relatively free. In these cases neurological signs are variable, but there is almost never complete paralysis as if the cord were involved

In the presence of massive herniations either centrally or laterally several

though it is certainly rare.

Clinical Picture.—Having discussed the pathological aspects of degenerative disc disease it is possible to fit the clinical picture into some semblance of order First of all there is the underlying progressive process of degeneration which proceeds silently but may become symptomatic in one of several ways:

1 By mechanical derangement of the intervertebral joint it may lead to instability which in turn predisposes to ligamentous or muscular strain

2 A sudden extrusion or protrusion may lead to nerve root involvement with severe sciatica.

3. Both may develop simultaneously or either one follow the other These are complicated cases from the diagnostic and therapeutic points of view

4. Occasionally the process will proceed slowly and never seriously

joint disease)

the section on
strain Nothing need be added here. There are no especial clinical findings to distinguish this type of case from others producing instability (spondylolysis, abnormal facets, etc.).

The classical case of extrusion or protrusion is difficult to miss. The patient is very often an otherwise perfectly healthy man in the middle age group (thirty to fifty) who if he has had any trouble at all before will have only had a mild backache.
(trivial or severe) in which the ba
is heard or felt and pain is experie
a severe sciatica develops with numbness and paresthesias. The patient

bending to the side of the sciatica. Straight leg raising is markedly restricted on the involved side and often this leg cannot be straightened out. The patient prefers the sitting or flexed position to any other. There is marked paravertebral tenderness with reproduction of sciatica on percussion or deep palpation.

Neurological signs are present. The variability of these has already been described. Usually the only one detected is that of reflex weakness or loss; occasionally there is slight sensory disturbance or motor weakness The accuracy of localization by neurological finding varies tremendously. It is certainly more accurate, when there are motor or sensory signs However, since one disc may involve either of two nerve roots the level of the lesion may still defy accurate localization.

Diagnosis of the level of the lesion is made as accurately as possible by neurologic findings, level of tenderness and *x*-ray changes if present In this latter respect a P.A. film of the lumbar spine in right and left lateral bending may show the level by restriction of motion in one intervertebral disc.[7] A myelogram is not usually necessary.

In the presence of a classical case of this sort, surgery will usually be advisable Since 95 per cent of disc herniations occur at the L4–L5 and L5–S1 interspaces, these two joints should be explored unless there is evidence of pathology at a higher level. This can be done without weakening the structure of the back appreciably. On the whole, fusion is not necessary in these cases. It is true that there is chance of further trouble (instability and strain) later, but the frequency of this does not seem
The
from
ssical
case is dramatic unless nerve root pressure has been present for a prolonged period.

A trial of conservative treatment may be tried if the patient so wishes
packs are ad-
The degree of
tinue unabated
and straight leg raising does not improve in a period of three weeks, operation is definitely indicated.

Relief of pain from conservative therapy may follow absorption of the disc material, or pressure on the nerve root may cause cessation of conduction with paralysis Because of the considerable overlap of innervation in the lower extremities, paralysis of one nerve root will usually lead to surprisingly little loss of function. If pain is relieved conservatively the

patient should increase his activities gradually and be instructed in postural principles and exercise.

It is in the **complicated cases** that most difficulties of diagnosis and treatment arise. In most of these cases there is a combination of the symptoms [illegible]
Since the
other low
logical and laboratory analysis

Treatment must be attuned to the individual case. Specialist care is advisable In the majority of cases a favorable response will result from conservative therapy. On the other hand, some will need surgery At the present state of knowledge, there is no satisfactory clinical method of differentiating one case from another Surgical indications vary from one

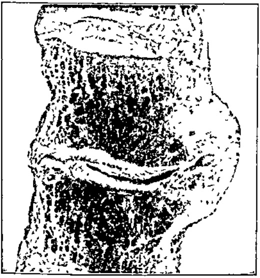


FIG 386 —A longitudinal section of the spine showing an old stage in degeneration with narrowing of the disc and spurring anteriorly

orthopedist to the next, and are conditioned largely by their individual experiences Suffice it to say, there is nothing to be lost by a trial of conservative therapy. There is no group of cases in which the old adage of elective surgery is truer; let the patient make the decision for surgery [25]

Conservative therapy is again based on general principles of rest and posture. These have been described fully elsewhere and do not need to be repeated A surgical approach will usually mean a combined operation of nerve root exploration, decompression, and spinal fusion. Convalescence is slow. It demands the fullest attention and patience of both the surgeon and patient.

The phase of **secondary osteoarthritis** is usually seen in the older age groups By the fifth decade it is not uncommon to find extensive spurring at the anterior margins of the vertebral bodies. In applying the diagnosis

of osteoarthritis to the spine, however, we are far more concerned with

completely worn away with eburnation of the underlying bone. The synovial lining of the capsule *often is fibrosed and thickened* with slight villous processes. The inflammatory changes are minimal At the junction of the capsule with the facet osteophytic formation is often present.



FIG 387 —X-rays to show an old stage of degenerative disc disease The disturbance of spinal mechanics has accelerated osteoarthritic changes in the facets.

Osteoarthritic changes in the spine as elsewhere may give *surprisingly* little pain. The typical complaints are *stiffness* and mild aching pain noticed particularly at night or in the early morning and improving *with* mild exercise. However, because of the loss of movement and of the resiliency of the spine, these patients are more prone to injury than normal individuals The presence of osteoarthritis alone is not enough to explain an episode of *acute* back pain Something more has been added Ligaments may have been strained in consequence of movement of the joints beyond the limits of their contracture Spurs may have been fractured *and even more* extensive changes may take place.

Sciatica in the presence of osteoarthritis also implies more than spinal [illegible] of the apophyseal joints may encroach on [illegible] sure. Narrow- [illegible] ner narrows the [illegible] brae may project posteriorly and implicate a nerve root in much the same way as a disc herniation Actual protrusion of degenerated disc remnants may occur.

Treatment of these cases in the acute phase follows the principles of rest, sedation and heat. Rest may be obtained by recumbency, bracing

or strapping depending on the severity of complaints. In cases with nerve root pain traction may be of some help As the symptoms subside the patient is instructed in a gentle routine of exercise to mobilize his joints gradually and to improve his posture. He is instructed how to use his back so as to avoid excessive strain. Prolonged use of a brace is avoided whenever possible.

In those cases that seek aid in the chronic phase of symptoms, gentle exercise, salicylates and occasionally the use of a support are indicated [illegible]

exacerbation of pain is not relieved by adequate conservative management Orthopedic opinion should be obtained prior to consideration of surgery

V. OTHER CONDITIONS PRODUCING LOW BACK PAIN

Infections.—1 It is not uncommon for backache to occur as part of the toxemia of a generalized infection In these cases diagnosis will usually not be a problem Except for muscle soreness, there are few if any localizing signs More important is the back pain that accompanies meningeal irritation There is usually in these cases a generalized spasm of the erector spinae which may be detected by Kernig's maneuver or by limitation of straight leg raising. When severe, as in infectious meningitis, it will produce opisthotonus. It is important to remember that back pain with muscle tightness is one of the early signs of anterior poliomyelitis

2 Infections may cause backache by direct involvement of spinal struc- [illegible]

localization to the spine by abscess formation. As in long bones, *x*-ray changes do not occur until approximately two weeks after onset Since most of the organisms encountered (Staphylococcus, most commonly) are penicillin sensitive, the course of this illness is often curtailed by its administration. However, whenever possible, the organism should be cultured and its sensitivity to the various chemotherapeutic agents determined A blood culture is usually the source, but if a soft tissue abscess presents itself, aspiration should be attempted, unless incision and drainage is planned

Occasionally an acute infection will settle in the disc itself. Again the symptoms and signs will be those of a severe febrile illness In these cases, the exact localization may not become evident for months when *x*-rays will finally show narrowing or obliteration of the disc space *Under proper* chemotherapy the illness will long since have subsided.

3 Perhaps of a greater interest in the differential diagnosis are the granulomatous infections By far the most important of these is tuberculosis, though brucellosis and fungus (particularly coccidiomycosis) infections also occur

Pott's disease (*tuberculosis*) is extremely variable in its symptomatology due to the marked individual variation of resistance. Back pain may be present for several months before other characteristic changes of the disease occur. Systemic signs, particularly otherwise *unexplained weight loss,* should make one suspicious. Night pain, night cries, fever, previous pul-

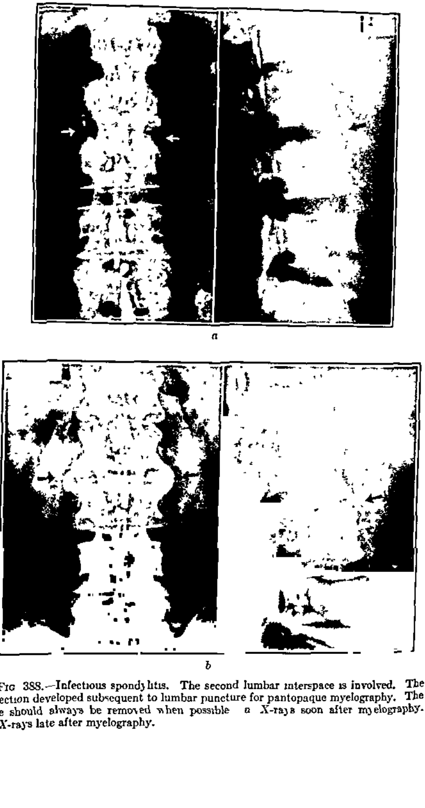


FIG 388.—Infectious spondylitis. The second lumbar interspace is involved. The infection developed subsequent to lumbar puncture for pantopaque myelography. The dye should always be removed when possible *a* X-rays soon after myelography. *b* X-rays late after myelography.

typical changes.

It is important to remember that this disease often involves the sacroiliac joints Here again x-ray evidence of bone destruction is the most important diagnostic finding.



a b

FIG 389 —Tuberculosis *a* Tuberculous spondylitis Note abscess (arrow) There is destruction of the 6th dorsal intervertebral disc space This case is too high to cause pain in the lower back. When involvement is in the lumbar spine, the abscesses frequently follow the plane of the psoas muscle and x-rays will show distention of the muscle border *b* Sacroiliac tuberculosis Note sclerosis and irregularity of right sacroiliac joint

4 *Herpes Zoster.*—Infection of the dorsal lumbar root ganglions by the herpes zoster virus may cause severe sciatica. Early diagnosis is difficult,

complicated by severe cord or root damage since there is often accompanying subluxation or dislocation. In the presence of neurological changes it is wise to refer these types of injury immediately to medical centers where they can receive the combined care of various groups of specialists

2. Fractures of the spinous processes are rare in the lumbar spine However, it is not uncommon to find fractures of one or more transverse proc-

esses. This group of cases usually responds well to supportive treatment (*i.e.*, rest, sedation and strapping) Sometimes injection of the injured area with novocaine provides remarkable relief.

3 Fractures of the sacrum usually result from direct trauma Localized tenderness and superficial hematomata help to make the diagnosis. These fractures are difficult to demonstrate by *x*-ray. Fractures of the pelvic ring are often associated with *dislocation of the sacroiliac joints. Proper* *x*-ray evaluation is not difficult

4 *Pathologic Fracture.*—Because of the great forces brought to bear upon the spine, it is not surprising that collapse of the bony structure often occurs when there has been weakening due to destruction or lack of bone repair Not infrequently the pain of the fracture will be the first indication of the pathologic process. Lack of adequate trauma should cause one to suspect this diagnosis. The following lesions are those most likely to lead to *collapse* of a vertebra:

a. Metastatic Malignancy
  1 Breast, kidney, thyroid and lung particularly in the adult.
  2 Neuroblastoma in children
  3 Unknown. It is not uncommon for the primary lesion to defy diagnosis.

b Primary Neoplasms
  1 Hemangioma.
  2. Multiple myeloma.
  3. Giant Cell Tumor

c. Metabolic Dyscrasias
  1. Senile Osteoporosis.
  2 Osteomalacia. The adult form of Vitamin D deficiency is rare in this country
  3. Hyperparathyroidism.

d. Tuberculosis

e. Conditions of Uncertain Etiology
  1. Gaucher's disease.
  2 Eosinophilic Granuloma.

Differential diagnosis of these conditions depends on a complete history and physical examination and full use of laboratory and radiographic techniques Biopsy of the lesion itself should be done whenever possible. [illegible] blood vessels

laboratory and radiographic picture is sufficiently conclusive in itself. These patients are sometimes markedly debilitated.

Rheumatic Diseases.—Rheumatoid Spondylitis.—Of primary importance is rheumatoid spondylitis (Marie Strumpell's arthritis, spondylose rhizomelique, ankylosing spondylitis) This syndrome is described in greater detail in the chapter on Spondylitis, page 547 Suffice it to say that it is [illegible] checked

and by the characteristic *x*-ray changes (obliteration of the sacroiliac joints and calcification of the spinal ligaments). It is most important to

prevent deformity once the condition is recognized and while treatment is being instituted. X-ray therapy is often dramatic in providing relief from pain.

2. **Osteoarthritis** (see p. 720).—The sacroiliac joints very often show degenerative changes that may be responsible for symptoms Sclerosis of bone adjacent to the joints is seen in the x-ray. Treatment consists of

re exact
tis, very
osteoid
dition is

porosis) is a definite and troublesome syndrome that is commonly associated with back pain.

The etiology of this condition is not definitely known There is suggestive experimental and clinical evidence of its relation to endocrine disturbances, and clinically treatment with estrogenic and androgenic hormones has proved successful[1] Pathogenically speaking the spine is weakened, presumably due to lack of new bone formation to replace that continually lost in the natural catabolic processes. That this is the whole story is doubtful since senile osteoporosis certainly shows a predilection for the spine and though the extremities may be involved, the skull almost never is. Pain only occurs when there is mechanical insufficiency of bone and not infrequently as the result of pathologic compression fractures

Though the condition is most frequently encountered in women in the postmenopausal state, particularly following an induced cessation of ovarian function, it is seen sometimes in young women and even in men One is usually struck by the elderly appearance of the patient Typically

frequently the condition is associated with collapse of one or more vertebræ The laboratory findings are variable and depend largely on careful metabolic control of the patient during the time of study The serum calcium and alkaline phosphatase are normal, but the phosphorus may be slightly elevated. A Sulkowitch test may show increased urinary calcium excretion.

Treatment must combine the alleviation of pain and the correction of the underlying metabolic fault. In the former instance, the principles to be follow

Bed re

can be

and th

should be determined and if achlorhydria is found it should be corrected since it interferes with calcium absorption.

Under therapy symptomatic relief is usually gratifying. However, improvement by *x*-ray except for healing of fractures is disappointing. In many of the milder cases *x*-ray changes in response to treatment do not seem to occur. This is undoubtedly related to the fact that moderate



Fig. 390.—Senile osteoporosis Note washed out appearance of vertebræ with ballooning of discs and partial collapse of D12 and L1.

changes in bone substance may take place without their being apparent radiographically.

2. ***Osteomalacia.***—*In the adult skeleton where growth has ceased,* vitamin D deficiency causes a very similar picture to that of senile osteoporosis Due to the lack of calcium absorption and increased urinary phosphate excretion, the mineral substance of bone is rapidly lost and

weakness results. This condition is fortunately rare in civilized areas, unless there is some related disease such as sprue interfering with absorption of the fat-soluble vitamins. The changes are more widespread than the average case of senile osteoporosis and careful laboratory analysis will lead to diagnosis.

3 **Hyperparathyroidism** —Osteitis Fibrosa Cystica, as hyperparathyroid changes in the skeleton are termed, leads to a clinical picture similar to the two previously mentioned syndromes Here the fault is thought to be one of increased urinary phosphate excretion with consequent destruction of bone to maintain the ionic mineral balance in the tissue



FIG. 391 —Paget's disease of right innominate bone Note sclerosis, enlargement and coarsening of trabecular structure Osteogenic sarcoma may supervene

4 **Paget's Disease** —This peculiar affliction of bone not infrequently involves the pelvis and occasionally presents itself in the spine. Confusion with senile osteoporosis, osteomalacia and hyperparathyroidism is not likely, since the *x*-ray changes are quite characteristic The trabecular pattern is coarsened, the bone hypertrophies, and there is loss of the denser lines of stress. Osteoporosis may be marked. The condition is often asymptomatic and when causing pain the possibility of a superimposed osteogenic sarcoma should not be forgotten The serum alkaline phosphatase is often markedly elevated.

Neoplastic Disease.—Keeping in mind that any of the connective tissue neoplasms are possible there are certain tumors that show a predilection for the spine.

1. *Intraspinal Lesions*.—There are very few men experienced in spinal surgery who have not at one time or another encountered an intraspinal tumor causing backache and sciatica. Recently a report from the Mayo Clinic[5] has shown the incidence of various lesions. Neurofibroma and ependymoma each occurred thirteen times in over a thousand cases. Carcinoma metastatic to the spinal cord and its supporting structures occurs as well as other primary tumors. The important fact, however, is that these conditions develop quite frequently in the *absence* of neurologic signs. Pantopaque examination is often necessary to detect these lesions.

2. *Lesions of Vertebral Column*.—*a*. Of the malignant tumors by far the most common are *multiple myeloma* and *metastatic carcinoma*. Both often have a very similar *x*-ray appearance and can lead to surprisingly extensive bone destruction. Both are often widespread. Differentiation depends on the demonstration of a primary source (prostate, breast, kidney, thyroid and lung most frequently) in the case of metastatic disease and of an



FIG. 392.—Spinal cord tumor. Note widening of interpedicular diameter and flattening of pedicles (arrow). There is pressure erosion of the posterior portions of the vertebra in the lateral view.

elevated total protein and plasma globulin in the case of multiple myeloma. Bence-Jones protein when present is positive evidence of the latter. Biopsy often is necessary to distinguish between these two conditions. Of rare occurrence in the malignant group is chordoma.[4] This lesion is largely confined to the sacrococcygeal and cranial portions of the axial skeleton. It arises from remnants of the primitive notochord, and is locally invasive. It is quite resistant to both surgical and roentgen therapy.

b. Benign tumors of the spine are rather uncommon. Giant cell tumor, bone cysts, osteochondromas and chondromas may occur, but hemangiomata and osteoid osteomas deserve special consideration. It is probably wrong to class all hemangiomata as benign since some lead to quite extensive bone destruction. However, many of these tumors respond very favorably to irradiation. The *x*-ray picture when typical is easily recognized. There are vertical striations in the vertebral body and this

often partially crushed so as to be broadened in all dimensions but the vertical Those hemangiomata that behave more malignantly very often involve the posterior elements and form large paraspinal masses with scattered calcific deposits

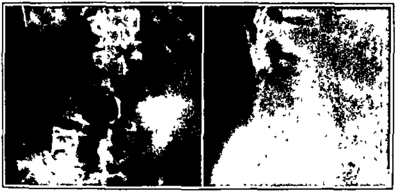


FIG 393 —Multiple myeloma The 3rd lumbar vertebra is completely destroyed There is rarefaction and partial destruction of L2 and L4 The associated neurologic changes were surprisingly few

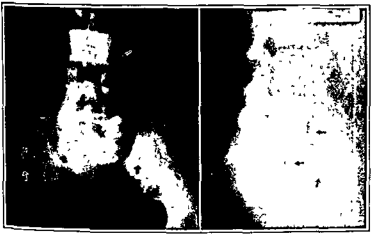


FIG 394 —Prostatic carcinoma with scattered osteoblastic metastases (arrow)

Osteoid osteoma is a tumor but relatively recently recognized [12] A small focus of ... urrounding
s ... en difficult
to ... a zone of
dense bone with a small nidus of decreased density more or less in the center. The pain may be severe and often occurs at night. It is so frequently relieved by aspirin that this is used as a helpful diagnostic test at the Hospital for Special Surgery.

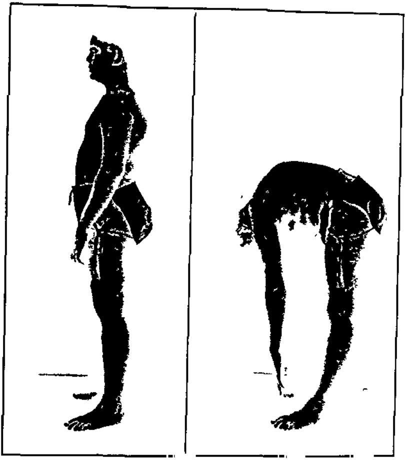


FIG 398 —Dorsal round back The photographs show the abnormal dorsal curve and the deformity is accentuated by forward bending

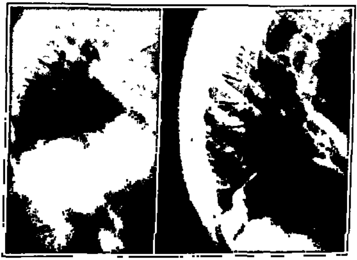


FIG 399 —X-rays of dorsal round back. There is anterior wedging of several of the dorsal vertebræ in the x-rays which were made four years apart

Scoliosis.—A lateral curvature of itself very rarely, if ever, causes pain However, it may lead to degenerative changes and thus become symptomatic. In this situation treatment of the scoliosis *per se* is not usually necessary.

Coccygodynia.—Pain in the coccygeal area deserves individual consideration since it is often troublesome in diagnosis and treatment Coccygeal pain may be of three separate types.

1. *Primary Coccygeal Abnormality* —As the result of a direct trauma or childbirth, the coccyx may be fractured or the sacrococcygeal joint severely strained.

2 *Primary Low Back Pathology* —It is not uncommon for coccygodynia to develop as a secondary phenomenon in a patient with low back pain The mechanism is not understood, but the pain is thought to be of the referred type.

3 *Visceral Lesion.*—It is not uncommon for patients with rectal or genitourinary disease to develop coccygeal pain This is of a referred type and sometimes associated with "spasm" of the muscles of the pelvic floor It is only by careful evaluation that these three entities may be separated The back must always be examined since trauma to the coccyx cannot usually occur without simultaneously involving the low back Rectal examination should include palpation of pelvic structures as well as analysis of the coccyx and its adjacent structures (*i e*, the coccygeus and levator ani muscles, sacrospinous and sacrotuberous ligaments)

Proctological, gynecological and urological evaluation may be indicated In those cases where examination points to primary pathology in the sacro-coccygeal articulation, coccygectomy may be effective if conservative therapy fails Every effort should be made to avoid irritation of the painful area. Soft chairs are usually worse than hard and the patient should sit with good posture. A rubber ring may be necessary Sometimes a strapping or "sacro-iliac belt" will be helpful by pulling fatty tissues over the non-padded coccygeal region. Massage and diathermy may provide symptomatic relief

sacral plexus.

Pain from Hip Joint Disease.—Occasionally it is quite difficult to distinguish the pain of hip disease from that of the spine, since both may radiate along the same pathways Intra-articular disease often causes pain radiating down the inner aspects of the thigh to the knee, whereas the pain of a trochanteric bursitis may follow the more usual "sciatic" distribution For this reason it is most important to analyze hip joint movements in all cases with sciatica and especially those in which the findings in the lower back are eq

Referred Pain from Visceral Structures.—It is imperative to remember that a lesion in one of the pelvic organs or the retroperitoneal structures can cause pain referred to the back. In women, the most usual condition is one of a retroverted uterus, but disease of the ovaries and tubes may also be at fault. The pain in these cases tends to be worse during menses. In men, referred low back pain is usually the result of a prostatic lesion. Stones within the urinary tract are easily diagnosed in the presence of typical ureteral colic, but at other times may be less evident. In the presence of associated anorexia and weight loss a retroperitoneal neoplasm must always be considered.

In most such cases the back findings are insufficient to explain the symptoms. In elderly patients, however, there may be associated minor lower back lesions which make diagnosis more difficult. Not infrequently these patients will be considered psychoneurotic, and proper therapy delayed. In the case of a malignant neoplasm, time may be needlessly lost These cases can be most difficult to evaluate and more than any other, will test the diagnostic acumen of the attending physician.

BIBLIOGRAPHY

1. [illegible]
2. [illegible]
3. BUCHMAN, J · Arch Surg, 34, 23, 1937.
4. COLEY, B. L [illegible]
5. CRAIG, SVIE [illegible]
6. DANFORTH, [illegible] 5
7. DUNCAN, W [illegible]
8. EHRENHAFT [illegible]
9. HARE, H. F [illegible]
10. HITCHCOCK, [illegible]
11. INMAN, V [illegible]
12. JAFFE, H. L [illegible]
13. KELLGREN, [illegible]
14. ——— Clin Science, 4, 35, 1939
15. KUHNS, J G, *et al* Posture and Its Relationship to Orthopedic Disabilities, [illegible]
16. [illegible]
17. [illegible] 211, 210, 1934
18. [illegible] Gynec and Obstet, 66, 288, 1938.
19. [illegible]
20. [illegible] and INMAN, V T Arch Surg, 40, 383, 1940
21. [illegible] Joint Surg, 32, 841, 1950.
22. [illegible] 387, 1944.
23. [illegible] Hygiene, 4, 106, 1922.
24. [illegible] 74, 270, 1947.
25. [illegible] Course Lectures, Vol 9, 53,
26. [illegible] 1952

INDEX

A

Achillodynia, 1012
Acromegalic arthritis, x-ray changes in, 95
Acromegaly, 220
  joints in, 694
Acromioclavicular joint, examination of, 34
    injection of, 331
    x-ray changes in hyperparathyroidism, 95
Acrosclerosis, 677, 678
Acrylic prosthesis, 383, 384
ACS, 247
ACTH (*See also* Corticotropin), 288–317, 523
  in gout, 748, 764
  in lupus erythmatosus, 665
  in psoriatic arthritis, 170
  in Reiter's syndrome, 811
  in rheumatoid arthritis, 241
  in rheumatic fever, 605–609
  in sarcoidosis, 889
  with gold therapy, 284
Actinomycosis, 851
  x-ray changes in joints, 94
Adaptation syndrome, 524
Addison's disease, 523, 524
Adenofibroma, 895
Adhesions, development of, 132
Adhesive plaster splinting, 343
    strapping of ankle, 915
      of knee, 915
      of low back, 917
      of wrist, 915
Adiposis dolorosa, 218
Adrenal corticoids (*See also* Cortisone), 288–317
  glands and arthritis, 523–525
  hormones, ascorbic acid function, 545
    excretion, 192
  physiology, 524
Adrenals, fetal, 533
  in rheumatoid arthritis, 180
Adrenocortical steroids, 289
Adventitious bursae, 145
A/G ratio in rheumatoid arthritis, 191
Agglutination tests, 187–188
Aging of joints, 691–703
Ainhum, 874, 1016
Alarm reaction, 524
Alkaptonuria, 775–776
  x-ray appearance of bones, 95
Allergic rheumatism, 626
Allergy in rheumatic fever, 584, 585
Amyloid degeneration, 180–182
Analgesics, 238
Anatomy of articular structures, 42, 48, 130–131, 1024–1031
Anemia, from gold therapy, 270
  in rheumatoid arthritis, 187
Angioneural arthrosis, 626
Ankle, examination of, 38
  hydrocortisone injection technic, 328
  prevention of deformity, 354–356
Ankylosing spondylitis (*See* Rheumatoid Spondylitis), 547–570
Ankylosis, 29
  development of, 132
Anterior metatarsalgia, 1010
Anthrax, 851
Antibiotics, for "L" organisms, 512
  in infectious arthritis, 844–846
  in rheumatoid arthritis, 247
  in rheumatic fever, 614
Anticoagulants in treatment, 539–540
Antifibrinolysins, 189, 588
Antihistaminics in hydrarthrosis, 633
Antihyaluronidase, titers in rheumatic fever, 589
Antimalarials, 665
Antimony poisoning, 874
Anti-reticular cytotoxic serum, 247
Antistreptolysin test, 189
Antistreptolysins, 588
[illegible]
[illegible]
[illegible]
[illegible]
  703
Apophyseal joint changes, 562
Arachnodactylia, 221
Arch supports, 994–997
Army arthritis centers, 24
Arneth-Schilling, 186
Arsenicals, in rheumatoid arthritis, 248
Arthralgia, menopausal, 694
Arthritis and Rheumatism Foundation, 21
  atrophic (*See* Rheumatoid Arthritis)
  classification of, 16–18
  clinic, organization of, 118–121
    staff needed, 118–120
  definition, 15
  deformans (*See* Degenerative Joint Disease), 691
  degenerative (*See* Degenerative Joint Disease), 691
  differential diagnosis, 199
    by x-ray, 64–106

Arthritis, examination of patient, 25–41
gonococcal, 779–791 (See Gonorrheal *Arthritis)*
hemophilic, 877–883
hypertrophic, 691 (*See* Degenerative Joint Disease)
in tropical diseases, 866–876
Ainhum, 874
big heel, 874
bacterial diseases, 867–869
bacillary dysentery, 867
bubonic plague, 868
cholera, 868
epidemic tropical acute polyarthritis, 868, 869
leprosy, 867, 868
tularemia, 868
epidemic dropsy, 874
fungus diseases, 872, 873
Madura foot, 872, 873
sporotrichosis, 873
helminth injections, 873, 874, 875
cysticercosis, 873
dracontiasis, 873
filariasis, 873
loiasis, 873
onchocerciasis, 873
trichinosis, 873
lathyrisus, 874
protozoal diseases, 870, 871
leishmaniasis, 870
malaria, 870, 871
trypanosomiasis, 870
pyomyositis, 874
rickettsial and allied diseases, 871, 872
oroya fever, 872
Q fever, 871
Rocky Mt spotted fever, 871
trench fever, 871
typhus, 871
schistosomiasis, 874
spirochetal diseases, 869, 870
bejel, 869
relapsing fever, 870
Weil's disease (infectious jaundice), 869
yaws, 869, 870
tropical ulcer, 874
viral diseases, 871
dengue, 871
psittacosis, 871
Rift Valley fever, 871
sand-fly fever, 871
yellow fever, 871
vitamin deficiency diseases, beriberi, 872
osteomalacia, 872
pellagra, 872
scurvy, 872
incidence, 21–24
...t, *1018*
Arthritis, osteoarthritis, 691 (*See* Degenerative Joint Disease)
periodicals, 120–121
physical signs of, 28–40
post scarletinal, 202
prevention of deformities, 337–368
relative incidence of, 21–24
rheumatoid, 123–494 (*See* Rheumatoid Arthritis)
treatment of, 224–250
senescent, 691. (*See* Degenerative Joint Disease)
symptoms of, 25–27
traumatic, 907–932
Arthrodesis, 380
cartilage, changes in rheumatoid arthritis, 136
function of, 44
structure of, 43
jelling, 26
resections, 380
Artisone (acetoxypregnenolone), 248
Aschoff bodies, 172
Ascorbic acid, 545
Aseptic necrosis, 693, 921
Ashburn General Hospital, 24
Aspidium poisoning, 874
Aspiration of synovial fluid, 54, 325
Aspirin, 238
Atabrine, use in treatment, 665
Atophen, 768
Atrophic arthritis (*See* Rheumatoid Arthritis)
spondylitis (See Rheumatoid Spondylitis), 547, 570
Atrophy, muscular, 158, 159
Sudeck's, 921
Atypical rheumatoid arthritis, 160, 199
Aureomycin, 845, 846
Axioms of Hench, 759

B

BACILLARY dysentery, 851, 866, 867
and Reiter's syndrome, 795
Back, anatomy of, 1024–1031
braces for, 395–397
examination of, 35, 36
pain, 1023–1080
from hip disease, 1079
from visceral disease, 1080
medical history, 1031
physical examination, 1032–1043
x-ray examination, 1044–1046
Backache, 1023–1080
industrial, 917

Backstrain, 1046–1059
  pathogenesis, 1048
  posture, 1048
Bacterial endocarditis, 220
Baker's cysts, 925
Bakers, luminous, 409, 410
BAL, in gold toxicity, 275
Balanitis circinata, 803, 804
Balneotherapy, 491
Bamboo spine, 74, 552, 562
Barometric changes, 517
Basal metabolism, in rheumatic arthritis, 191
Baseball finger, 712
  pitcher's shoulder, 970
Bed exercises in arthritis, 340
  rest, 230
    positions, 337
Bee venom, 246
Bejel, 869
Benemid, 748, 768
Benign lymphogranulomatosis, 885
Beriberi, 872
Big heel, 874
Bismuth, in scleroderma, 681
Black water fever, 871
Blastomycosis, 851, 1015
Blood chemistry, in rheumatoid arthritis, 190
  count, in rheumatoid *arthritis, 186*
  culture in rheumatic arthritis, 191
  dyscrasias, from gold therapy, 270
  transfusions, 242
    pregnant donors for, 536
Boeck's sarcoid, 885 (*See* Sarcoidosis)
Bogomolets serum, 247
Bone absorption, 70
  acute atrophy, 921
  caisson disease, 921
  density, 65
  eburnated, 703
  exostoses, 703
  metabolic diseases *of, 1071–1073*
  spurs, 703
Bony enlargement at joints, 29
  spurs, 1012
Bornholm disease, 645
Bouchard's nodes, 32
Bougainville rheumatism, 868
Brachial neuralgia, 715–718
Brachydactylia, 162
British Anti-Lewisite, 275
Bronchitis, from gold therapy, 270
Bronzing of skin, 155
Brucellosis, 847–850
  spondylitis, *x-ray appearance of, 92*
Bs, use in treatment, 546
Bubonic plague, 868
Buck's extension, 344, 347
Buerger's disease, 217
Bunion, *1009*
Bursa, hygromatous, 144, 145
Bursa, adventitious, 145
  lesions in rheumatoid arthritis, 144–146
  tumors of, 901
Bursitis, calcific, *x-ray* findings, 105, 110, 111
    x-ray treatment for, 110, 111
  calcareous, 945
  cortisone and ACTH in, 301
  ischial, 908
  prepatellar, 908
  radio-humeral, 908
  subacromial, 945–960
  subdeltoid, 908, 945–960
  syphilitic, 843
Butazolidin, 764 (*See* Phenylbutazone)
  in rheumatoid arthritis, 239
Butterfly rash, in lupus, 663

C

CAISSON disease, 921
Calcaneal epiphysitis, 1020
  spurs, 94
Calcareous deposits about shoulder, 105, 110, 111
Calcification in subdeltoid bursitis, 952
  of spinal ligaments, 74
  of supraspinatus tendon, 945
Calcified bursitis, 945
Calcinosis, 685–690, 758
  circumscripta, 685
  clinical picture, 687
  diagnosis, 688
  disturbed physiology, 686
  etiology, 685
  pathogenesis, 687
  treatment, 688
  x-ray appearance, 105, 106, 679
Calcium deposits, 105
  gout, 685 (*See* Calcinosis)
Caliper splints, 389
Camptocormia, 504
Capillary resistance, in rheumatic fever, 589
Capsulotomy, posterior, 377, 379
Carcinoma, metastatic, 892, 1074
Cardiac involvement, 597–598
  lesions in lupus, 662
    in rheumatoid arthritis, 171–176
Carpo-metacarpal joints, injection of, 334
Cartilage, hyaline, 43
  physiology of, 44
  reparative ability, 692
  semilunar, 934, 936
Castex splints, 348
Causalgia, 921, 1014
Cell counts, synovial fluid, 52
Cells of synovial fluid, 51
Cellular changes in synovium, 135
Celluloid splints, 348
Cellulose acetate splints, 348

Cervical fibrositis, 644
  rib syndrome, 965–970
  spine, examination of, 35
Chalk gout, 106, 685, 758 (*See* Calcinosis)
Charcot joint (neurogenic arthropathy), 837
Charcot's joints in spine, 571
  x-ray appearance of, 90–92, 93
Chaulmoogra oil, 247
Chilling, 517
Chloromycetin, 845, 846
Chloroquin, 665
Cholera, 868
Cholesterol, in rheumatic fever, 589
Chondroitin sulfuric acid, 123
Chondroma, 894
Chondromalacia of patella, 939
Chondromyxomas, 894
Chordoma, 1074
Chorea, 590–592
Chorionic gonadotropins, 534, 537
Chrysotherapy, in rheumatoid arthritis, 241, 251–286
Cinchophen, 768
Circulation in joints, 46
Clammy hands, 155
Classification of arthritis, 16–18
Claudication, intermittent, 219
Clicking jaw, 35
Climate and rheumatic diseases, 517–521
  in prophylaxis, 616
  in rheumatic fever, 580–581

-121

Club foot, 1016
Cobra venom, 247
Coccidioidomycosis, *x*-ray appearance of joints, 94
Coccidiosis, 851, 853
Coccygodynia, 1079
Colchicine, in acute gout, 762–764
  in chronic gout, *769*
  in intervals of gout, 768
Cold air, 429
  as causative factor, 517
  compresses, 429
  contrast baths, 429
  use in arthritis, 428
Collagen, chemistry of, 651
  diseases, 649–690
    cortisone for, 308–309
    fibrositis as, 640
    dermatomyositis, 681–685
    disseminated lupus, 659–666
    gold therapy in, *284*
    lupus erythematosus, 659–666
    pathogenesis, 652
    periarteritis nodosa, 655–659
    rheumatic fever and, 654
    rheumatoid arthritis and, 653
Collagen diseases, scleroderma group, 669–681
    similarities, 649
    summary of features, 650
    undifferentiated, 650
  embryology of, 651
  pathology of, 651
Colloidal gold, 258, 280
Community Rehabilitation Center, 479–480
Compound E, 288–317 (*See* Cortisone)
Compound F, 319–336 (*See also* Hydrocortisone)
Congenital deformities about the foot, 1016
Conjunctivitis, 801
Connective tissue, diseases of, 649–690
    ground substance, 123, 140, 649
Contractures, 159
  of joint capsule, 29
Contrast baths, 423–424
Convalescent hospital, 480
Copper salts, 241
Cord serum, 536
Coronary occlusion, in rheumatic fever, 587
Corpus luteum, 533
Corticoid excretion, 525
Corticoids, chemistry of, 289
Corticotrophic hormone, in degenerative joint disease, 731
Corticotropin, 288–317, 523
  anti-inflammatory effect, 294
  contraindications to use, 311
  in gold toxicity, 277
  in gout, 748, 764
  in rheumatic fever, 300, 605–609
      adverse effects, 607
      contraindications, 606
      dosage, 607
      indications, 605
      length of treatment, 609
  in rheumatoid arthritis, 305
      spondylitis, 569
  indications for use, 310
  manner of action, 298
  metabolic effects, 291
  pharmacologic actions, 290
  preparations of, 296
  prevention of complications, 312
  use in diffuse collagen diseases, 308–309
  with gold therapy, 284
Cortisone, 288–317
  actions of, 290
  anti-allergic action, 293
  anti-inflammatory effects, *294*
  anti-rheumatic effect, 297
  chemistry of, 289
  clinical uses for, 297
  complications, 306
  contraindications to use, 311
  cytologic effects, 293
  dangers from use, 299
  effects on central nervous system, 293

Cortisone effects on infections, 299
endocrine effects, 293
enzymatic changes from, 293
in bursitis, 301
in collagen diseases, 309
in degenerative joint disease, 731
in dermatomyositis, 685
in fibrositis, 308
in gold toxicity, 277
in gout, 301, 748, 764
in lupus erythematosus, 665
in ochronotic arthritis, 776
in osteoarthritis, 307
in psoriatic arthritis, 170
in Reiter's syndrome, 811
in rheumatic fever, 300
in rheumatoid arthritis, 241, 301–307
spondylitis, 568–569
in sarcoidosis, 889
in scleroderma, 681
indications for use, 310
insulin with, 316
limitations, 309
maintenance therapy, 305
manner of action, 298
metabolic effects, 291
physiologic side effects, 299
post-treatment problems, 314
preparations of, 295
prevention of complications, 312
suspension, 295
with gold therapy, 284
with para-aminobenzoic acid, 316
Costen's syndrome, 918
Costovertebral joints, 36
Course of rheumatoid arthritis, 158
Coxa vara, 723
Cracking of joints, 29
Creatinuria, in scleroderma, 678
Crepitation, types of, 29
Criteria for therapeutic results, 249
Cuboid wedge, 1001
Curare, 350
Curative workshop, 480
Cushing's syndrome, 299, 523, 524
Cystic degeneration of nodules, 146
Cysticercosis, 873
Cysts, Baker's, 925
detritus, 703
in bone ends, 83
of semilunar cartilages, 936
popliteal, 925

D

DACTYLITIS, syphilitic, 832
Dampness, 517
Deficiency diseases, 872
Deformities, correction of, 370–403
operations for, 375–386
prevention of, 337–368
Deformity, in physical examination, 29
Degenerative arthritis, 691 (*See* Degenerative Joint Disease)
changes in aponeuroses, 702, 703
in ligaments, 702–703
in tendons, 702–703
joint disease, 691–737
age, 708
and menopause, 694
aseptic necrosis, 692, 693
climate, 519, 708
differential diagnosis, 205
effusions in, 705, 713
endocrine disturbance, 694
estrogen therapy, 529
etiology, 691–694
focal infection, 692–693, 734
Heberden's nodes, 711–712, 734
heredity, 694
history, 691
incidence, 708
joints involved, 709
laboratory findings, 709–710
of cervical spine, 715–718, 734
of hip joint, 721–725, 735, 736
of knees 712–714, 736
of lumbar spine, 720, 735
of rib joints, 725
of sacro-iliac joints, 720
of spine, 573–574, 714–721
roentgenographic findings, 573–574
treatment of, 575–576
of thoracic spine, 718–720, 735
orthopedic management, 385–388
pathogenesis, 694, 703–705
pathological features, 703–706
physiologic stress 692
roentgenographic appearance, 710
sex, 708
symptoms, 708–710
synovial fluid in, 53
membrane in, 700
treatment, 728–737
corticotrophic hormone, 731
cortisone, 731
diet, 730
drugs, 730–734
general considerations, 728–730
hydrocortisone, 731
iontophoresis, 734
of specific lesions, 734
phenylbutazone, 731
physical therapy, 729–730
rest, 728
roentgen therapy, 733
vitamins, 733
vascular factors, 693
x-ray findings in, 80–84
Delta-5-pregnenolone, 248
Dengue, 871
Depolymerization of hyaluronate, 123

Depression, mental, 160
DeQuervain's disease, 923
physical signs of, 34
Dermatitis, from gold salts, 268
Dermatomes, 1030
Dermatomyositis, 681–685
clinical features, 681–684
cortisone for, 309
differential diagnosis, 684
laboratory findings, 684
pathology, 681
treatment, 684–685
Dermatophytosis, 1015
Desoxycorticosterone, 524–525
Devil's grip, 645
Diabetes, from cortisone, 299, 313
Diarthrodial joints, rheumatoid lesions in, 131
Diarthroses, 42, 130
Diathermy, 412–417
advantages of each type, 416
contraindications to local use, 417
in malum coxae senilis, 453
long wave, 413
medical, 416
microwave, 415
precautionary measures, 414
short wave, 414
wave lengths and frequencies, *413*
Dicoumarol, 539
Diet, in gout, 766–767
in rheumatoid arthritis, 237, 542–546
Differential diagnosis, 199–222
fibrositis *vs* psychogenic ·
507
gout *vs* rheumatoid arthr
rheumatoid arthritis *vs*
joint disease, 207
vs fibrositis, 209
vs gonorrheal arthritis, 203
vs rheumatic fever, 203
sheep cell agglutination, 149
Differentiation of nodules, rheumatic fever, 141, 154
rheumatoid arthritis, 141, 154
Diffuse collagen diseases, cortisone for, 308–309 (*See also* Collagen Diseases)
Diphtheria, 853
Disc, intervertebral, degeneration of, 1059–1067
pathogenesis, 1060–1063
symptomatology, 1063–1064
treatment, 1064–1065
Dislocations of spine, 1069
Disseminated lupus erythematosus, 659–666
after rheumatic fever, 593
course, 660
differential diagnosis, 214, 664
etiology, 659
features, summary, 660
laboratory findings, 663
Disseminated lupus erpthematosus, L cells, 663
pathology, 659
physical findings, 661–663
summary of features, 660
symptomatology, 660
treatment, 665–666
Distribution of joint involvement, 26
DOCA, 248, 523
Dracontiasis, 873
Drug intoxication, 219
Duplay's disease, 945
Dupuytren's contracture, 977–981
etiology, 977–978
pathology, 978
symptoms, 978–979
treatment, 978–981

E

Eburnation of bone, 83
Edema, from cortisone, 299, 312
Effleurage, 431
Effusion of joints, signs of, 29
Elbow, arthroplasty, 383–384
correction of deformity, 400
examination of, 34
hydrocortisone injection into, *332*
prevention of deformity, 362
Electric blanket, 412
cabinet baths, 411
Endocrine disturbance in degenerative joint disease, 694
factors in rheumatic disease, 522–530
theory, 125
Eosinophilia, in gold therapy, 270
Ependymoma, 1074
Epidemic acute tropical polyarthritis, *868–869*
dropsy, 874
Epiphyseal bone changes in rheumatoid arthritis, 136–138
eburnation, 138
granulations, 137
osteoporosis, 137
Epiphysitis, 1020, 1077
Episodic rheumatoid arthritis, 628
Epithelioid cells, 143
Ergopyrine, 239
Erosion of bone, 137
of cartilage in rheumatoid arthritis, 136
Ertron, 245, 545
Erysipelas, 853
Erythema annulare rheumaticum, 600
marginatum, 587

Erythema, multiforme, 600
nodosum, 590, 600, 666, 667
cortisone for, 308
Erythematodes, 659 (*See also* Disseminated Lupus)
Erythralgia, 218
Erythrocyte sedimentation, 56–62
Erythromelalgia, 218
Estrin therapy, 520
Estrogens, 529–537
in degenerative joint disease, 733
Etiology of rheumatoid arthritis, 123–127 (*See under each type*)
Euphoria, from cortisone, 293
Ewing's tumor, 898
Examination of arthritic patient, 25–41
of back, 1032–1043
of joints, 30–40
of fingers, 30–33
Exercise, 443–454
for bed patients, 340
summary, 236
therapeutic, 445–453
active, 445
assistive, 445
bimanual, 447
for patient in bed, 449
head traction, 452, 453
hydrogymnastics, 446
in acute arthritis, 452
in arthritis of knees, 453
in calcified tendinitis, 454
in cervical arthritis, 452
in chronic arthritis, 452
in degenerative spondylitis, 453
in fibrositis, 451
in malum coxae senilis, 453
in rheumatoid spondylitis, 452
in subdeltoid bursitis, 454
isometric contraction, 445
list of exercises, 450–451
manipulation, 446
medical motion pictures, 454
mobilization, 446
muscle setting, 445–447
passive, 445
performance, 447
postural, 452
prescription for, 448
progressive resistance, 448–449
pulley weights, 454
resistive (active), 445
use of crutches, 453
with help of apparatus, 446
Exercise-rest balance, 444
Exostoses, multiple, 891
on spine, 593
Eye lesions, 182–183, 801–803

F

Fango, 493
Faradism, 459
Feet, arches of, 984
examination of, 987, 988
flat, 989–998
arch supports, 994–997
congenital, 1016
signs, 992
symptoms, 991
treatment, 993, 994
of advanced type, 1007
normal physiology of the, 984–986
painful, 983–1021
achillodynia, 1012
anterior metatarsalgia, 1010
arthritis, 1018
bony spurs, 1012
calcaneal epiphysitis, 1020
circulatory disturbances, 1016–1017
classification, 983
congenital deformities, 1016
epiphysitis, 1020
flat feet, 989–998
Freiberg's disease, 1021
hallux rigidus, 1010
valgus, 1009
hammer toe, 1009
inflammatory lesions, 1015–1016
immersion foot, 1013–1015
Kohler's disease, 1020
march foot, 1019
metatarsal arch, depression of, 1008–1009
metatarsus varus, 1010
overlapping toes, 1011
painful heel, 1012
pes cavus, 1012
plantar fasciitis, 1019
trench foot, 1013–1015
tumors, 1016
weak, 989, 998
adhesive strapping, 1005
exercises and postural aid, 1003, 1004
Felty's syndrome, 162, 186
Fever therapy, 245, 456–457
contraindications, 457
in Reiter's syndrome, 809
physiological and chemical effects, 456
Fibrillation of cartilage, in rheumatoid arthritis, 136
Fibrinoid change, 154
swelling, in nodules, 140
Fibroblasts, in rheumatoid nodules, 140
effect of cortisone on, 294
Fibroma, 895
Fibrosarcoma, 898
Fibrositis, 635–647
bursal, 636
cervical, 641
classification, 635
climatic factors, 517
cortisone for, 308
differential diagnosis, 645
effect of jaundice, 538

Fibrositis, etiology of, 637
exposure, 638
infection, 637
occupation, 637
psychological factors, 638
tension, 638
trauma, 637
exercises in, 454
incidence of, 636
intramuscular, 636
laboratory findings, 642
myalgic spots, 640
nodules in, 641
panniculitis, 636
pathology of, 641, 642
periarticular, 636

secondary, 636
symptoms, 643
tendinous, 636
treatment of, 646, 647
trigger areas, 640
versus psychogenic rheumatism, 499, 507, 638–639
Filariasis, 873
Fingers, examination of, 30–33
Finkelstein test, 34
First metatarsal segment, deficiency of, 997, 998
Fish vertebrae, 528
Flail joints, 33
Flat feet, 989–1000
Flexion contractures,
Floating patella, 37
Focal infection, 509–516
and degenerative joint disease, 692, 693, 734
arthritis, 211
as possible cause of rheumatoid arthritis, 124
treatment of, 515

examination of,
injection of joints, 328
prevention of deformity, 354–356
strain, predisposing causes of, 984
Foreign protein therapy, 244
Foxhole arthritis, 868
Fractures of spine, 1069
of tibial spine, 941
osteochondral, 941
Freiberg's disease, 1021
Friction effect, 43
Frozen shoulder, cortisone for, 308 (See also under Shoulder)
Function of joints, 42–47
Functional complaints, 497–508
Fungus diseases, 872–873
infections of the foot, 1015
Fusiform swelling, 151

G

GALL bladder, changes in rheumatoid arthritis, 179
Galvanic currents, 457, 458
Gamma globulin, 191, 650
Ganglion, 926
with rheumatoid arthritis, 144

Genu valgum, 712
varum, 712
Giant cell tumors, 1074
Glanders, 853
Glass arm, 944, 970
Globulins, placental, 534
Glomerulonephritis, 592
Glomus tumors, 903
Glucocorticoids, 534
Glucose tolerance, in rheumatoid arthritis, 191
Glycine, 769
Gold bronchitis, 270
dermatitis, 268
salt therapy, 241, 251–286
types of, 280
sodium thiosulfate, 257, 280
therapy, benefits, 285
clinical experience, 261
contraindications, 283
controlled studies, 263
dosage, 282
effect of toxicity on arthritis, 277
excretion of gold, 255
gastrointestinal toxicity, 269
history of, 251, 286
in animals, 252
in degenerative joint disease, 734
laboratory investigation, 252–261
limitations of, 285
metabolism of gold, 254–259
method, 279–283
mode of action, 259
mouth lesions from, 269
nephritis, from, 269
precautions, 274
relapse following, 278
resumption of, after toxicity, 278
selection of cases for, 266
technique, 282

Gold therapy, therapeutic effects, 265
  toxicity, 268–274
    incidence of, 270
  treatment of complications, 275
 toxicity, 268, 274
  pathogenesis, 271
  prevention of, 273
  treatment of, 275
 with cortisone, 284
Gonadotropic hormone, 526
Gonadotropins, 534
Goniometer, 34, 444, 474
Gonococcal myositis, suppurative, 786, 787
Gonorrhea, as precipitating cause for rheumatoid arthritis, 515
 with rheumatoid arthritis, 782, 783, 784
Gonorrheal arthritis, 202, 779, 793
  affecting multiple joints, 780
  age incidence, 780
  analgesics, 789, 790
  aspiration of affected joints, 790
  associated features in, 787, 788
    acute hemorrhagic glomerulonephritis, 787
    bacteremia, 788
    cutaneous manifestations, 787
    keratodermia blennorrhagicum, 787
  clinical course, 782
  complement fixation test, 785
  criteria for cure, 790
  diagnosis, 779, 784, 785
  differential diagnosis, 783, 784, 785
  fever therapy, 790
  general care, 790
  gonococcus carriers, 788
  incidence, 779
  incubation period, 780
  joint fluid examination, 785
  laboratory tests, 784, 785
  pathology, 780, 781
  penicillin, 788, 789, 790
  penicillin resistant gonococci, 789, 790, 791
  rest and prevention of deformity, 786
  roentgenographic changes, 785, 786
  skin tests, 785
  spurs on os calcis, 94, 785
  streptomycin, 788, 789
  sulfonamides, 788, 789
  suppurative myositis, 786, 787
  tenosynovitis, 785, 786
  treatment, 788, 789, 790
  versus Reiter's syndrome, 784
  x-ray changes in, 93, 94
Gout, 739, 770
 ACTH for, 764
 alcohol in, 762
 allergy as factor, 744
 Benemid for, 768
 calcium, 685 (*See* Calcinosis)
 chronic gouty arthritis, 755
 cinchophen in, 768
 climatic factors in, 519
 clinical picture, 753, 759
 colchicine for, 762, 764, 768
 cortisone for, 764
 course, 753, 755
 definition, 739
 diagnosis, 758
 diet in, 766–768
 differential diagnosis, 204, 754
 electrolyte changes, 750
 etiology of, 741–744
 heredity in, 741, 744
 history of, 740
 hyperuricemia, 748
  ACTH effect on, 748
  Benemid effect on, 748
  cortisone effect on, 748
  mechanism of, 744, 746
  occurrence of, 745
  significance of, 743
 incidence of, 740
 interval treatment, 766
 intra-articular hydrocortisone in, 766
 laboratory findings, 758, 759
 low 17-ketosteroids, 750
 ocular inflammations, 758
 pathogenesis of, 743
 pathology of, 750–753
 predisposing factors, 740, 742
 prognosis, 770
 provocative factors, 761
 renal complications, 756
 roentgen findings, 752, 760
 salicylates in, 769
 tophi, 754, 755, 770
 trauma in, 762
 treatment of, 762, 770
  acute attack, 762, 776
  chronic arthritis, 769–770
  intervals, 766–769
 uric acid diuretics, 768
   excretion, 747, 750
    Benemid effect on, 748
    corticotropin effect on, 748
    cortisone effect on, 748
   metabolism, 745, 750
   renal excretion, 747
 vascular factors, 743
 x-ray differential diagnosis, 88
  findings in, 102–105
Gouty arthritis, cortisone in, 304
  synovial fluid in, 53
Grades of improvement, 249
Granulations in rheumatoid arthritis, 136
 of epiphyseal bones, 137
Growing pains, 597

H

Hallux rigidus, 1010
 valgus, 1009

Hammer toe, 1009
Hand, correction of deformity, 401
  prevention of deformities, 363, 365
Hands, examination of, 30–33
  causalgic states, 32
  degenerative joint disease, 32
  rheumatoid arthritis, 31
  scleroderma, 32
Haverhill fever, 853
Heart murmurs, 598
Heat, application of, 408
    dry heat, 409
    moist heat, 418–428
      paraffin, 421
  bubble baths, 422
  cabinet baths, 411
  conduction, 406
  contrast baths, 423
  conversive, 406
  diathermy, 412, 417
  effects of local application, 408
    of systemic application, 408
  electric blanket, 412
  foam baths, 422
  fomentations, 418
  full wet pack, 422
  heating jacket, 411
  hot air douche, 411
  hot tub bath, 420, 421
  hydrotherapy, 420, 428
  infrared lamp, 410
    radiation, 412
  Hubbard tank, 425–427
  lamps, 409
  luminous baker, 409
  paraffin applications, 420
  radiant, 406
  therapeutic pools, 428
  tolerance of patient, 410
  ultrasonic radiation, 416, 418
  value in arthritis, 409
  whirlpool bath, 424
Heberden's nodes, 711
    differential diagnosis, 210
    physical signs, 32
    x-ray appearance of, *82*, *85*
Heel, painful, 1012
Heliotherapy, 454, 456
Helminth infections, 873, 875
Hemangioma, 896–898, 1074, 1076
Hemarthrosis, 879, 880, 910
  treatment of acute, 882
    of chronic, 882
Hematologic disorders from gold therapy,
  270

Herpes zoster, 219, 1060
High frequency (oudin) current, 458
Hilton's law, 45
Hip, acrylic prosthesis, 383, 384
  correction of deformity, 392–395
  examination of, 36
  hydrocortisone injection of, 329
  motions of, 1037
  obturator neurectomy, 387
  osteoarthritis of, orthopedic management, 386–388
  osteotomy, 379
  prevention of deformity, 357
Hippocratic finger, 216
Histoplasmosis, 854, 855
  x-ray changes in joints, 94
History of arthritis, *15*
History taking in arthritis, 25–27
    course, 27
    distribution of joint involvement, 26
    family history, 25
    joint pain, 26
    joint swelling, 27
    past history, 25
    predisposing factors, *26*
    prodromes, 26
    types of onset, 26
Hormone excretion in rheumatoid arthritis, 192
Hormones, adrenal, 523, 525 (*See also under* Cortisone, etc)
  in gout, 743
  in treatment of scleroderma, *680*
  ovarian, 526–529, 536
  parathyroid, 529
  pituitary, 522, 523
  steroid, 525
  thyroid, 526
Hot air devices, 411, 412
    douche, 411
  fomentations, *418*, *419*
  paraffin applications, 420, 421
  tub baths, 420, 423
Hothouse palms, 155
Humidity, 517
Hubbard tank, 425–427
    resistive exercises in, 427
    use in arthritis, 427
    water temperature, *426*
Hyaluronic acid, 123

629–633

16, 319–336

Hydrocortisone, contraindications to local use, 322
indications for local use, 321
intra-articular doses, 335
for degenerative joint disease, 731
for intermittent hydrarthrosis, 633
for sprains, 913
in gout, 766
injection, 319-336
local use of, 316, 319-336
physiologic effects, 294
preparations of, 296
technic for intra-articular use, 325-336
use in orthopedics, 376
Hydrotherapy, 420-428, 491
bubble bath, 422
contrast baths, 423
foam bath, 422
hot tub bath, 420, 421
Hubbard tank, 425-427
technique of applying paraffin, 420
therapeutic pools, 428
wet pack, 422
whirlpool bath, 424
Hygromatous bursæ, 145
Hyper-cortisonism, 299
Hyperparathyroidism, 530, 1073
x-ray changes in acromioclavicular joints, 95
Hypersensitivity, as cause of rheumatoid arthritis, 511
Hyperthyroidism, 526
Hypertrophic arthritis (*See* Degenerative Joint Disease), 691-736
pulmonary osteoarthropathy, 215-217, 526
spondylitis, 572-576
Hypertrophy of synovial membrane in rheumatoid arthritis, 132
Hyperuricemia, significance of, 743
Hypoparathyroidism, 530
Hypopituitarism and arthritis, 522
Hypothyroid rheumatism, 526
Hysteria, 505

I

Iliac compression test, 1042
Incidence of rheumatic diseases, 21
Industrial backache, 917
Infection, focal, 509-516
of joints, as cause of rheumatoid arthritis, 511
Infections while under cortisone, 314
Infectious arthritis, 842, 865 (*See also under* individual infections)
arthritis with ulcerative colitis, 850, 851
aureomycin, 845, 846
brucellosis, 847-850
diagnosis, 848, 849
Infections brucellosis, joint involvement, 849, 850
laboratory tests, 848
treatment, 850
chloromycetin, 845, 846
diagnosis, 842, 843, 844
miscellaneous forms, actinomycosis, 851
anthrax, 851
bacillary dysentery, 851, 866, 867
blastomycosis, 851
coccidioidal arthritis, 851, 852, 853
diphtheria, 853
erysipelas, 853
glanders, 853
Haverhill fever, 853
histoplasmosis, 854, 855
infectious hepatitis, 851
infectious mononucleosis, 855
influenza, 855
lymphogranuloma venereum, 855, 856
measles, 856
meningococcal arthritis, 856, 857, 858
mumps, 858
paratyphoid fever, 859
puerperal septicemia, 856
rat bite fever, 853, 854
scarlet fever, 858
smallpox, 858, 859
spirochetemia, 859
subacute bacterial endocarditis, 859
typhoid fever, 859
orthopedic management, 846, 847
penicillin, 844, 845, 846
pharmaceutical arthritis, 860
prognosis, 844
roentgenographic findings, 843, 844
streptomycin, 844, 845, 846
symptoms, 842, 843
terramycin, 845, 846
treatment, 844, 845, 846
jaundice, 851, 860
hepatitis, 851
mononucleosis, 855
spondylitis, 576
theory, 124
Inflammation in muscle, 141
Inflammatory lesions of the foot, 1015, 1016
Influenza, 855
Immersion foot, 1013-1015
Innervation of joints, 44-46
Insulin, use with cortisone, 316
Integument, lesions in rheumatoid arthritis, 143
Interfibrillar substance, 123
Intermittent hydrarthrosis, 629-633
differential diagnosis, 631
etiology, 629
pathology, 631

Intermittent hydrathrosis, prognosis, 632
  symptoms, 629
  treatment, 632
Internal derangement of knee, 933–942
Interstitial calcinosis, 685, 688
Intervertebral discs, degenerative changes, 704
  herniation of, 1059, 1067
Intra-articular hydrocortisone, 319–336
Intramuscular fibrositis, 636
Intravenous trypsin, 540
Iodides, in rheumatoid arthritis, 248
Ion transfer (Iontophoresis), 458, 459
Iontophoresis, for degenerative joint disease, 734
Iridocyclitis, 182
Iritis, 182
Irrigation in acute subdeltoid bursitis, 954
Isotopes in gout study, 748

J

Jaundice, effect on arthritis, 538, 540
  following transfusion, 242
  in rheumatic diseases, 538–540
Jaw, examination, 35
Jelling, articular, 26
Joint fluid, 48–54 (*See* Synovial Fluid)
  manipulation, 372
  pain, types of, 26
  paracentesis, technique of, 325–336
  potentials, 47
  temperature, 43, 45, 46, 47
Joints, aging, 694–703
  examination of, 30–39
  infections, 919
  innervation of, 44–46
  involved in rheumatoid arthritis, 148
  loose bodies in, 703
  mice, 736
  surgery in degenerative joint disease, 736
  tumors of, 890, 905
  villi in, 697, 700, 703, 705
  wounds, 919
Jugular compression test, 1041
Juvenile rheumatoid arthritis, 161, *162*
  cortisone in, 307
  gold therapy for, 268
  x-ray findings in, 71
Juxta-articular adiposis dolorosa, 218

K

Ketosteroid excretion, 525
Ketosteroids in scleroderma, 678
Keratoconjunctivitis sicca, *162*
Keratodermia blennorrhagicum, 787, 800, 804, 805
Keratosis blennorrhagica, 801, 804, 805
Kidney changes in rheumatoid arthritis, 179
Knee, braces for, 389, 392
  correction of deformity, 388, 392
  degenerative joint disease, 712, 736
  examination of, 37
  hydrocortisone injection technique, 328
  internal derangements, 933, 943
  loose bodies, 939
  nylon arthroplasty of, 382, 384
  osteoarthritis of, orthopedic *management, 386, 388*
  posterior capsulotomy, 377, 379
  prevention of deformity, 351, 354
  soft tissue lesions, 941
  synovectomy of, 375
  traumatic synovitis, 916
Kohler's disease, 1020
Kyphosis, 1034
  dorsal, adolescent, 1077

L

Laboratory findings, in degenerative joint *disease, 709–710*
  tests in work-up, 40
Lacrymal secretions, 163
Lactic acid, intra-articular, 320
LaMain en lorgnette, 97, 99
Laseque test, 1041
Lathyrism, 874
Lauron, 254, 280
L E cells, 663, 664
Legg-Calvé-Perthes disease, 723
Leishmaniasis, 870
Leprosy, 867, 868
Lesions in joints (*See under* specific disease or pathology)
Leukemia, 217
  infantile, *vs* rheumatic fever, 592–593
Leukopenia, 187
Libman-Sacks endocarditis, 662
Ligaments, cruciate, 937
  degenerative changes, 702, 703
  lateral, 937
Liman, 493
Limitation of joint motion, 29
Lipohemarthrosis, 910
Lipoma, 218, 895
Liposarcoma, 898
Liver function in rheumatoid arthritis, 191
  involvement, and etiology, 123
  pathology, 177
  toxicity, from gold therapy, 269
Loiasis, 873
Loose bodies in knee, x-ray appearance, 97, 100
"L" organisms, 512, 797
Lover's heel, 785

Low back strain, 1046–1059
  frequency currents, 457–459
    faradism, 457
    galvanism, 457
    sinusoidal current, 458
Lumbago, 644
Lumbosacral strain, 1046–1059
  pathogenesis, 1048
  symptomatology, 1053–1057
  treatment, 1054, 1057, 1059
Lupus erythematosus, cortisone for, 308
  disseminated, 659–666 (*See also under* Disseminated Lupus)
  pernio, 885
Lymph nodes in rheumatoid arthritis, 179
Lymphogranuloma venereum, 213, 855, 856

M

MADURA foot, 872, 873, 1015
Malaria, 870, 871
Malignant synovioma, 898
Malta fever, 847, 850 (*See* Brucellosis)
Malum coxae senilis, 691, 721, 735
    x-ray appearance of, 81, 84
Manipulation of arthritic joints, 372, 373
  of joints, legal responsibility, 446
March foot, 1019
Marie-Strumpell's disease, 547, 570 (*See* Rheumatoid Spondylitis)
Massage in arthritis, 429, 443
  compression, 431
  contraindications, 442
  effleurage, 431, 432
  general regimen for patient, 439
  home treatment, 443
  in chronic stage, 441
  indications for, 442
  kneading, 437
  mechanical vibrations, 438
  medical motion pictures, 454
  movements used, 431
  percussion, 431, 438
  petrissage, 437
  physiological effects, 429, 431
  positioning of patient, 438
  prescription for, 433
  stroking, 431
  summary of technique, 439
  technique, 437
  therapeutic value, 442
  vibration, 432
Masson body, 587
MCP joints, 30
Measles, 856
Meningococcal arthritis, 856–858
Menopausal arthralgia, 694
  arthritis, 527
Menopause and arthritis, 526–529
  degenerative joint disease in, 694
  painful joints in, 694
  stress factors, 527
Mental reactions from cortisone, 293, 313
Mester test, 189
Metabolic bone disease, 1071–1073
Metacarpophalangeal joints, injection of, 334
Metallic taste, 260
Metastatic tumors, 891
Metatarsal arch, depression of, 1008, 1009
  bar, 1000
Metatarsalgia, 1010
Metatarsus varus, 1010
Methyl salicylate, 613
Microclimate, 519
Microwave diathermy, 415
  dielectric myvalex, 415
  effects, 416
  wave length and frequency, 413
Military problem of arthritis, 22
Milkman's syndrome, 221
Mineral waters, 488, 490
Mixed arthritis, 19
  at menopause, 527
  x-ray appearance, 84, 89
Morning stiffness, 26
Morphea, 669, 677
Morton's metatarsalgia, 1010
Mucin, synovial, 50
Mud baths, 493, 494
Multiple myeloma, 898, 1074
Mumps, 898
Muscle changes in rheumatoid arthritis, 141, 142
  herniation, 930
  injuries, 927–931
  lesions in rheumatoid arthritis, 192
  nodules, 141
  spasm, relief of, 350
  wasting, 159
Musician's shoulder, 944
Myalgic spots, 614
Mycetoma, 1015
Myelograms, 1046
Myeloma, multiple, 898, 1074
Myocarditis, 172, 599
Myochrysine, 251, 280
Myositis, with rheumatoid arthritis, 141
Mysteroids, 248, 315
Myxedema, 526

N

NAFFZIGER test, 1033
Nail changes, 156
  in psoriatic arthritis, 167
Narcotics, 240
Narrowing of joint space, 83
National Institute of Arthritis and Metabolic Diseases, 20
Neck, examination of, 35
Necrotizing vasculitis, 655 (*See* Periarteritis)

Neoplasms, of spine, 1073, 1076
Neostigmine, 217
Nephritis, and rheumatic fever, 592
  from gold therapy, 269
Nephrosclerosis, in gout, 756
Nerve distribution in joints, 44
  lesions in rheumatoid arthritis, 192
  roots, anatomy, 1026–1027
Nervous system, effects from cortisone 293
Neural nodules, 143
Neuralgia, brachial, 717
Neuritis, from gold therapy, 270
  peripheral, 215, 715
Neurofibroma, 895, 1074
Neurogenic arthropathy, syphilitic, 837 (*See* Charcot Joint)
Nodules, 30
  from rheumatic fever, 599
  rheumatoid, pathology of, 139, 141 (*See also under* Subcutaneous Nodules)
  subcutaneous, 154
Non-articular rheumatism, 635, 647 (*See also* Fibrositis)
Nylon arthroplasty of knee, 382, 384

O

Obesity, from cortisone, 312
Obturator neurectomy, 387
  signs, 89
Occupational therapy, 465–472
  bibliography of books, 471
  crafts, 465, 466
    adapted to joint mobilization, 467
    types of, 467
  definition, 465
  effects, 469
  in arthritis of elbow, 468
    of shoulder, 468
  in hand deformities, 469
  in the arthritic patient, 469
  music with, 471
  objectives, 466, 467
  prescription for, 466
  supervision of, 467
  use for lower extremities, 468
  workshop in general hospitals, 470
Ochronosis (ochronotic arthritis), 775, 776
  x-ray changes in, 95, 96
Ochronotic arthritis, 775
Oroya fever, 872
Orthopedic correction of deformities, 370–403
  surgery in arthritis, 373 388
Os calcis, spurs on, 94
Osgood-Schlatter's disease, 920
Osteitis fibrosa cystica, 1073
  tuberculosa, 885
Osteoarthritic spondylosis, 704
Osteoarthritis, 691, 737 (*See also* Degenerative Joint Disease)
  at menopause, 527
  cortisone in treatment, 307
  of spine, 572, 576
    x-ray appearance, 83, 86, 87
  operations for deformity, 386, 388
  orthopedic management, 385, 388
  traumatic, 907
  vitamin B_{12} in, 546
Osteochondroma, 891
Osteochondromatosis, of knee, 940
Osteoid formation, 137, 138
  osteoma, 1075, 1077
Osteomalacia, 872, 1072
Osteomyelitis, 219
Osteophytes, 83, 703, 704
Osteophytosis, 704
Osteoporosis, from cortisone, 299, 313
  in rheumatoid arthritis, 137
  of spine, 1071, 1072
  painful, 921
  post-menopausal, 527
  post-traumatic, 100, 102, 921
Osteotomy, 378
Otto pelvis, 723
Oudin treatment, 458
Ovarian suppression, from cortisone, 313
Overlapping toes, 1011
Oxycorticoids, 525
  excretion in rheumatoid arthritis, 192

P

PABA, 238
  in dermatomyositis, 684
  in lupus erythermatosus, 665

Palindromic rheumatism, etiology, 625
  laboratory findings, 624
  nodules, 624
  roentgenographic changes, 624
  treatment, 627
  versus rheumatoid arthritis, 626, 627
Palisading of cells, 140
Palmar fascia, 978
Pannus formation, 134, 135
Paracentesis of joints, technique, 325–336
Paralysis agitans, 218
Para-amino benzoate, 238
  in lupus, 665
Para-aminobenzoic acid with cortisone, 316
Paraspinal ligaments, 563
Parathyroid glands in arthritis, 529
Paratyphoid fever, 859
Pantol-C, 539
Parkinson's disease, 218
Parrot's pseudoparalysis, 831, 832
Passive extension test, 1042
Patella, chondromalacia, 940
  dislocation, recurrent, 941
Pathological synovial fluid, 51
Pathology, of rheumatoid arthritis, 130–146
  osteoarthritis, 694–706
  nodules, 139–141
  spondylitis, 551
Peats, 493
Pellagra, 872
Pellegrini-Stieda syndrome, 926
  x-ray appearance of, 96, 97
Peloids, 493
Pelotherapy, 493, 494
Penicillin, 844, 845, 846
  in rheumatic fever, 612, 615
  in rheumatoid arthritis, 247
Periarteritis, and rheumatic fever, 593
  nodosa, 655–659
  cortisone for, 309
  diagnosis, 657
  differential diagnosis, 214
  laboratory findings, 657
  pathogenesis, 656
  pathology, 655
  summary of features, 657
  symptomatology, 656
  treatment, 658
Periarthritis humeroscapularis, 945
  of the shoulder, 915
Periarticular edema, 29
  fibrositis, 200
Pericarditis, 172, 598, 601
Periostitis, syphilitic, 833
Peritendinitis crepitans, 927
Pes cavus, 1012
Petrissage, 437
Phenylbutazone, 239
  in degenerative joint disease, 731
  in gout, 764
Phlebitis, 1017
Phosphatase, 530
  alkaline, 220
Phosphorus, in rheumatic fever, 589
Physical examination in arthritis, 27
  management in treatment, 235
  medicine, definition, 405 (*See* Physical Therapy)
    in rheumatoid arthritis, 407
    prescription for, 406
  signs of joint disease, 28–40
  therapy, audio-visual aids, 480–482
    fever therapy, 456, 457
    heliotherapy, 454, 456
    heat, 406–428
    in arthritis, 405, 459
    in degenerative joint disease, 729, 730
    in home, 405
    ion transfer, 458, 459
    low frequency currents, 457, 459
    motion pictures, 481
    oudin treatment, 458
    precautions, 405
    rest and exercise, 443, 454
    types, 406–459
    written instructions, 406
Physiology of joints, 42–47
  of synovium, 49
PIP joints, 30
Pitting of nails, 156
Pituitary gland and arthritis, 523–525
  hormones (*See* Corticotropin)
  hypofunction, from cortisone, 299
  thyrotropic hormone, 523
Placental adrenocorticotropins, 534
  globulins, 534
  hormones, 533
  steroids, 534
Plantar fasciitis, 1019
Plasma transfusions in hemophilia, 881
Plaster splints, 336
Pleural effusion, 659
Pleuropneumonia-like organisms, 512, 797, 798
Pneumoroentgenography, 65
Poker spine, 563
Polyarteritis, 655 (*See also* Periarteritis Nodosa)
  acute, 150
Polycythemia vera, 217
Polyserositis, from lupus, 659
Poncet's disease, 827 (*See* tuberculous rheumatism)
Popliteal cysts, 925
Post-cortisone problems, 314
Post-menopausal osteoporosis, 527
Post-partum plasma, 536
Post-traumatic atrophy of bone, x-ray changes in, 100
Postscarletinal arthritis, 202
Posture, 338–343, 1043
Potassium deficiency, from cortisone, 299, 312

Pott's disease (*See* Tuberculosis of Spine), 1067–1069
PPLO, 797 (*See* Pleuropneumonia-like Organisms)
Precipitins, 189, 589

changes in, 534
products, therapeutic use, 535–538

-368

Primary fibrositis, 636
Problem of rheumatic disease, 19–21
Procaine injection for degenerative joint disease, 733
in subdeltoid bursitis, 956
in scalenus anticus syndrome, 969
intravenous, 734
technic, 913
for sprains, 913
intra-articular, 319

Prostate gland, 530
in rheumatoid arthritis, 180
Prostigmine, 247, 350
Protein changes, 191
Protruded disc, 1059–1067
Protrusio acetabuli, 723
Protozoal diseases, 870–871
Pseudo-appendicitis, 593
Psittacosis, 871
Psoriasis and arthritis, gold therapy, 268
and rheumatoid arthritis, 164–170
arthropathica, 164
pustular, 805
Psoriatic arthritis, 164–170
cortisone in, 307
course, 167
etiology, 166
treatment, 168
x-ray findings in, 80–82
Psychogenic factors, in fibrositis, 499–500
in rheumatism, 497–508
in rheumatoid arthritis, 497–499
rheumatism, 199
diagnosis of, 502–506
differentiation from fibrositis, 507
incidence, 501
psychopathology, 501
Psychoneurosis, 500
Psychosomatic factors, 497–508
rheumatism, 500–507

Psychotherapy in rheumatoid arthritis, 240
Public health problems of arthritis, 19–21
Puerperal septicemia, 858
Pulmonary lesions, in periarteritis, 657–658
of rheumatoid arthritis, 176–177
Punched out areas in bone, 89, 102
Purpura from gold therapy, 270
idiopathic, 667
rheumatica, 667
Pustular psoriasis, 805
Pyogenic arthritis, x-ray appearance of, 94
Pyomyositis, 874
Pyriformis syndrome, 1079

Q

Q fever, 871
Quinine, in lupus, 665

R

Radiculitis, cervical, 717
Radioactive substances in treatment, 113–114
Radiology in rheumatic disease, 64–114
tgen Changes, and

Raynaud's disease, 1017
phenomenon, 676–683
syndrome, 218
Reassurance, importance of, 22
Referred pain, 1030
Reflex dystrophy, 921
Rehabilitation, counsellor, 119
in orthopedic care, 402–403
of arthritic patient, 473–486
audio-visual aids, 480–482
basic needs, 473
cervical traction, 478
chair, high seat, 483
metal roller, 484
Community Rehabilitation Center, 479–480
convalescent hospital, 480
counterbalanced stairway chair, 482–483
curative work shop, 480
daily activity program, 475
test, 475
definition, 473
equipment, 475
for home care, 478–479
estimate of functional handicap, 474
gait re-education, 478
home care, 478–479
in conjunction with hormones, 478
in hospital, 475–476

progress record, 474–475
steps in physical rehabilitation, 477
time log (daily), 478
toilet seat, 484
use of apparatus, 477
of crutches and walker, 475–476
of physical medicine, 476
Reiter's syndrome, 794–811
clinical features, 805
pattern, 802–806
conjunctivitis, 801, 802, 803
course, 808
differential diagnosis, 806–808
from rheumatoid arthritis, 204
etiology, 795–798
genito-urinary symptoms, 802
joint pathology, 799
laboratory findings, 807
pathology, 798–802
pleuropneumonia-like organisms, 797
skin lesions, 800, 802, 803, 806
treatment, 808–811
Relapsing fever, 870
Relaxin, 533
Remission, definition of, 249
in rheumatoid arthritis, 159
spontaneous, 158
Renal lesions, in gout, 756
in rheumatoid arthritis, 179
Rest and exercise in arthritis, 443–454
abuse of rest, 444
effects of prolonged rest, 444–445
home treatment, 443
rest, 443–444
rest-exercise balance, 444
in rheumatoid arthritis, 229–232
of inflamed joints, 232
Rheumatic diseases, definition, 15
psychogenic factors in, 497–508
Rheumatic fever, 579–617
abdominal pain in, 593
allergy in, 584
and bacterial endocarditis, 590
and chorea, 590–592
and erythema nodosum, 590
and leukemia, 592
and lupus erythematosus, 593
and nephritis, 592
and cardiac involvement, 597–598
cardiovascular changes, 585–587
carditis in, 585
climatic factors, 518
corticotropin in, 300, 605–609
cortisone in, 300, 605–609
course, 601–604
hygienic factors, 581
hypersensitivity, 584
importance of, 579
incidence, 579
infections in, 583
laboratory findings, 588–590
pathogenesis, 583–585
pathology of, 585–588
predisposing factors, 580–583
prevention, 614–617
prodromes, 595
prognosis, 601–604
prophylaxis, 614–617
airborne infections, 615
climate, 616
penicillin, 615
sulfonamides, 614
public health and, 617
pulmonary changes, 587
complications, 600
recurrences, 599–601
relation to periarteritis, 593
to rheumatoid arthritis, 590
relationship to other diseases, 590–593
rest in bed, 609
serum reactions in, 589
skin lesions, 600
social factors, 581
streptococcal infection in, 583
symptoms of, 594–601
synovial fluid in, 53
tonsillectomy, 614
transmission of, 584
treatment, 605–614
ACTH in, 605–609
cortisone in, 605–609
general measures, 609–610
of congestive failure, 612
of joints, 613
salicylates in, 610–612
sulfonamides, 612
vitamin deficiency, 584
vs rheumatoid arthritis, 594
nodules, 599
peritonitis, 591
Rheumatism, definition, 15
psychogenic, 500–507
Rheumatoid arthritis, 123–286
adrenal hormone excretion, 192
amyloid degeneration in, 180–182
analgesics for, 238
and psoriasis, 164–170
antibiotics in, 217
atypical, 160, 199
blood chemistry, 190–191

Rheumatoid arthritis, blood studies, 186–190
butazolidin for, 239
cardiac lesions, 171–176
cause of death in, 183–185
climatic factors, *517–521*
clinical picture, 148–163
complications from cortisone, 306
correction of deformities, 370–403
cortisone for, 301–307
course of, 152–153, 158–160
diet in, 237, 542, 546
differential diagnosis, 199–222
effect of jaundice, 538
etiology of, 123–127
endocrine theory, 125
infectious theory, 124
initial lesion as clue, 123
neurogenic theory, *127*
nutritional deficiency theory, 127
psychosomatic theory, 126
sensitization theory, 125–126
stress and, 125
fever in, 153
focal infection, 509–516
gold salt therapy, 251–286 (*See also* Gold Therapy)
in children, 161–162
jaundice and, 538–540
laboratory findings in, 186–195
liver pathology in, 177
manipulation of joints, 372
mental factors, 497–499
mode of onset, 148–150
muscle lesions, 141
nail change in, 156
onset, 148–150
orthopedic operations, 373–388
pannus formation in, 135
pathology of, 130–146
articular lesions, 130–138
bursal lesions, 145
muscles, 141
nerves, 142
nodules, 139–141
skin lesions, 143
tendons and sheaths, 144–146
visceral lesions, 171–185
pattern of joint involvement, 148
peripheral nerve lesions, 142–143
physical management, 235
therapy in, 233–235
precipitation by gonorrhea, 515
pregnancy and, 532–538
pregnenolone in, 248, 315
prevention of deformity, 337–368
prognosis of, 160
progression of, 156
psychogenic factors in, 497–499
pulmonary lesions in, 177
remissions in, 158–159
rest in treatment, 229
Rheumatoid arthritis, *serological reactions*, 187–190
sheep cell agglutination, 188
skin changes in, 155
spinal cord changes, 144
splinting of affected joints, 343–350
symptomatology, 150–156
synovial fluid in, 51–53
systemic reactions, 149
treatment of, 224–250
anticoagulants in, 539–540
gold salts, 251–286 (*See also* Gold Therapy)
pregnancy blood, 536
hormones, 536–539
prevention of deformity, 337–368
treatment of summary, 229
trypsin, 540
with cortisone, 301–307
therapeutic criteria, 249
visceral lesions of, 171–185
vitamins in, 544–546
weight loss in, 154
x-ray differential diagnosis, 88
findings in, 67–71
nodules, 154
pathology of, *139–141*
significance of, 141–154
Rheumatoid spondylitis, 547–570
apophyseal joint changes, 562
as cause of low back pain, 1070
cervical involvement, 557
clinical course, 563
clinical picture, 553
cortisone in, 307
diagnosis, 564
etiology, 550
gold therapy, 268
incidence of, 550
laboratory findings, 558
ligamentous changes, 563
pathological changes, 138
pathology of, 557
phosphatase in, 530
pregnancy effects of, 533
sacroiliac involvement, 555
spinal changes, 555–557
symptoms of, 554
treatment, 566–570
cortisone and ACTH, 568–569
deformities, 566
estrogens in, 537
general measures, 566
orthopedic measures, 570
roentgen therapy, 107–110, 567
vs osteoarthritis, 565
x-ray findings, in, 72–80, 559
therapy for, 107–110
Rheumatologist, definition of, 118
Rheumatology, definition, 15
Rhizomelique spondylitis, 549
Rickets, 219

Rickettsial and allied diseases, 871–872
Rift Valley fever, 871
Rocky Mountain spotted fever, 871
Roentgen changes in rheumatoid arthritis, 193–195
therapy, 243, 567–568
for intermittent hydrarthrosis, 633
in rheumatic diseases, 106–114 (*See also* X-ray Therapy)
adverse effects of, 112
history of, 106
in bursitis, 110
in rheumatoid spondylitis, 107–110
rationale, 112
Roentgenographic differential diagnosis, 88
findings in arthritis, 64–106
Round back, 1077
Ruptured discs, 1059–1067

S

SACRO-ILIAC joints, anatomy, 1026
x-ray findings in rheumatoid spondylitis, 72
Sacro-iliitis, 564
Salicylates, in gout, 769
in rheumatic fever, 610
in rheumatoid arthritis, 238
toxic effects from, 611
Sand-fly fever, 871
Sanochrysine, 254, 280
Sarcoidosis, 885–889
clinical picture, 886
etiology, 885
laboratory findings, 887
pathology, 885
treatment, 888
x-ray appearance of bones, 95
Sayre head sling, 452
Scalenus anticus syndrome, 865–870
Scapulothoracic joint, 945
Scarlet fever, 858
Scheuerman's disease (dorsal kyphosis), 1077
Schilling count, 186
Schistosomiasis, 874
Schmorl's nodes, 1077
Schonlein's purpura, 667
Sciatica, 1023–1080
with spinal osteoarthritis, 1066
Scleredema, 670
Sclerodactylia, 676–677
Scleroderma, 669–681
clinical course, 675
cortisone for, 309
differential diagnosis, 678
etiology, 672–675
laboratory findings, 678
pathology, 670–672
physical signs, 677
Scleroderma, roentgen findings in, 678
symptoms, 676
treatment of, 680–681
with shoulder-hand syndrome, 675
Scoliosis, 1033–1079
Scotch douche, 429
Scurvy, 219, 872
Sedatives, 240
Sedimentation rate, 56–62
clinical interpretation, 60
in arthritis, 59–62
use in arthritis, 61
Westergren method, 58
Selye's hypothesis, 125
Semilunar cartilages, 934–936
cysts, 936
injuries of, 935
Senescent arthritis (*See* Degenerative)
Senile osteoporosis, 527, 1071
Sensation in joints, 45
Sensitized sheep cell agglutination, 188
Septic arthritis, synovial fluid in, 53
Serovasculitis, remittent, 659
Serum, *in pregnancy*, 534
sickness, 653–655
diagnosis, 655
pathogenesis, 655
symptomatology, 653–654
treatment, 655
17-ketosteroids, 525, 534
changes in, after *x*-ray therapy, 113
in gout, 750
in rheumatoid arthritis, 192
Sheep cell agglutination test, 188
Shoe corrections, 1000–1002
cuboid wedge, 1001
metatarsal bar, 1000
Thomas heel, 1000
Shoes, proper, 988–1002
Shoulder, correction of deformity, 398–400
examination of, 34
hydrocortisone injection technic, 341
painful, 944–975
causes of, 945
cervical rib syndrome, 965–970
chronic adhesive bursitis, 960–963
examination, 944–945
baseball pitchers, 970–971
rupture of supraspinatus tendon, 963–965
scalenus anticus syndrome, 965–970
shoulder-hand syndrome, 971–975
subdeltoid bursitis, 945–960
prevention of deformity, 358–362
Shoulder-hand syndrome, 971–975, 977
Sinusoidal current, 458
Sjögren's disease, 162
Skeletal traction, 370
Skin, changes in rheumatoid arthritis, 155
pathological changes in rheumatoid arthritis, 143
tests, streptococcal, 189

Trauma, micro, 907
Traumatic arthritis, 907–932
differential diagnosis, 212
pathological appearance, 909
primary, 908
secondary, 907
symptoms, 911
treatment, 911
x-ray findings in, 99–102
Treatment of rheumatoid arthritis, 224–250
of Still's disease, 162
Trench fever, 871
foot, 1013–1015
Trichinosis, 873
Trigger finger-tendinitis, 365
points, 644
Tropical disease arthritis (*See* Arthritis in Tropical Disease)
ulcer, 874
Trypanosomiasis, 870
Tryparsamide poisoning, 874
Trypsin, intravenous, 540
Tsutsugamushi fever, 871
Tuberculosis, diagnosis, 820
of knee joint, 820–821
of spine, 1067–1069
treatment, 821
Tuberculous arthritis, 813–830
aureomycin, 819
chloromycetin, 819
diagnosis, 814–815
differential diagnosis, 210
dihydrostreptomycin, 818
etiology, 815
isonicotinic acid hydrazide, 818–819
joints involved, 813
miscellaneous joints, 826–827
of finger, 826
of hip joint, 819
early symptoms, 819
treatment, 819
of shoulder, 826
of spine, 821–826
of spine pathology, 821–823
symptoms, 823
treatment, 823–826
para-amino salicylic acid (PAS), 818
pathology, 815–817
penicillin, 819
rheumatism, 827
roentgenographic findings, 815–816, 822–823, 825
streptomycin, 818
tenosynovitis, 827
terramycin, 819
treatment–general principles, 817–818
x-ray appearance in, 85–90
Tularemia, 868
Tumors of bursæ, 901
of feet, 1016
Tumors of joints and tendon sheaths, 890–905
chondroma and chondromyxomas, 94
classification, 891
diagnosis, 904–905
exostosis, multiple, 94
fibroma, 895
hemangioma, 896–898
lipoma, 895
malignant synovioma, 898
metastatic tumors, 898
osteochondroma, 891
xanthomatous giant cell tumor, 894
Types of joints, 130–131
Typhoid fever, 859
spine, 93
spondylitis, 93
vaccine, in rheumatoid spondylitis, 570
therapy, 244
Typhus, 871

U

[illegible] 850–851
[illegible]
chemical effects, 417
coarctation, 417
effects of vibrations, *416–417*
thermal effects, 417
technic of application, 418
Ultraviolet irradiation, 454–456
erythema reaction, 454
prescription for, 455
physiological effects, 455
therapeutic wave length, 455
types of lamps, 455–456
Undecylenic acid, 169
Underwater therapy, 491
Undifferentiated collagen disease, 650
Urate diuretics, 768–769
metabolism, 745–750
[illegible]
[illegible] drome), 794–811
Uveoparotid fever, 885, 887

V

VACCINES, 245
in degenerative joint disease, 734

Vascular changes in rheumatoid arthritis, 133
  lesions in rheumatoid arthritis, 180
Venoms, in hemophilic arthritis, 882
Vertebrae, anatomy, 1024–1031
  pathologic fractures of, 1070
Vesperal aching, 26
Villi, joint, 697, 700, 703, 705
Villous arthritis, 910
Viral diseases, 871
Virus infection, 511
Visceral lesions, rheumatoid arthritis, 171–185
Vitallium molds, 383
Vitamin A, 544
  B complex, 544
  B_{12}, 545
  C, 544–545
    with salicylates, 611
  D, 545
    and calcinosis, 689
    in rheumatoid arthritis, 245
  deficiency diseases, 872
    in rheumatic fever, 584
  E, 545
Vitamins, in scleroderma, 680
Vitiligo, 155, 679
Von Bechterew's syndrome, 549

W

Walking positions, proper, 1007
Weakness, 30
Weather, effect of on arthritic symptoms, 26
  effects on rheumatic disease, 517–521
Weber-Christian's disease, 645
Weight loss, 151
Weil's disease, 869
Weltman coagulation reaction, 189
Westergren technic, 58
Whirpool bath, 424–425
Wounds of joints, 919
Wrist, correction of deformity, 401
  hydrocortisone injection into, 331
  prevention of deformity, 363
Wrists, examination of, 33

X

Xanthomatous giant cell tumors, 894
X-ray differentiation, rheumatoid arthritis and gout, 88
    and osteoarthritis, 88
  findings in arthritis, 64–106
    in rheumatoid arthritis, 193–195
  of joints, dyes used in, 65
  17-ketosteroid changes from, 113
  technic in bursitis, 110
    in rheumatoid arthritis, 112
    in rheumatoid spondylitis, 107–109
  therapy (*See also* Roentgen Therapy), 106–114
    in spondylitis, 567–568
    side effects from, 568
  use in bursitis, 110–111
    in osteoarthritis, 112
    in rheumatoid arthritis, 112
    in rheumatoid spondylitis, 107–110

Y

Yaws, 869–870
  x-ray changes in, 93
Yellow fever, 871